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Antimicrobial Susceptibility Profile of *Escherichia Coli* Isolates from Urine Cultures in Patients that Attended the Regional Hospital Bamenda with Urinary Tract Infections

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ABSTRACT

Empirical antimicrobial therapy for urinary tract *Escherichia coli* infections in the Bamenda community and its environs where the Bamenda Regional Hospital is located, is of major importance since it has helped to obtain medications of choice, deduce appropriate prescriptions and a standard antibiotic profile for *Escherichia coli* in isolates from urine cultures obtained from patients.

This study was a prospective and analytical study on patients who consulted in the Bamenda Regional Hospital with one or more signs and symptoms of urinary tract infections. Adopting an open ended quantitative approach, we aimed at determining antimicrobial susceptibility profile for *Escherichia coli* from isolates of urine culture. Fifty (50) samples were considered in this study. The study precisely consisted of collection of specimens, identification and isolation of *Escherichia coli* strains in cultures, and conduction of in-vitro antimicrobial susceptibility testing by the Disc Diffusion Technique.

A total of 75 culture respondents were isolated using the CLED agar, out of these, 50 (66.7%) were *E. coli* positive against 9(33.3%) other bacteria . In the antibiotic susceptibility testing, there was 81% susceptibility to Ciprofloxacine, Cefotaxime (68%), Ceftriaxone (56%), Gentamycin (24%), Penicillin (18%), Doxycycline (12%), and Erythromycin (6%), Tetracycline (0%). Anti-bacterial susceptibility showed that ciprofloxacin (81%), was highly effective and to a lesser extent to the third generation cephalosporin (Céftriaxone 56% Céfotaxime(68%). High resistance was recorded for gentamycin (31%), Amoxicilline +A. clavulanique (36%), amoxicillin (50%), penicillin (50%), tetracyclin (62%), erythromycin (68%), doxycycline(81%). Traditional medicine and drug abuse may greatly contribute to drug resistance development in this environment.

This study challenges the health body as a whole to implement adequate means of epidemiological surveillance antibiotic sensitivity of bacterial strains isolated in laboratories.

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1. INTRODUCTION

The emergence and spread of antimicrobial susceptibility constitutes a major relieve in modern medicines. Resistance to antibiotics limits the success of antimicrobial agents in the therapy and prevention of infectious diseases. Yet society should be aware of the fact that many accomplishments of modern medicine have only been possible because of the availability of a protective antibiotic umbrella. However, continuous positive selection of resistant bacteria clones, whether pathogenic, commensal or even environmental bacteria, will modify the population structure of microbial communities, leading to accelerated evolutionary trends with unpredictable consequences for human health. Whatever the type of infection, treatment is based on the administration of antibiotics empirically (based on epidemiological data), or guided by the results of urine cytology examination (urine culture). Knowledge of empirical treatment failures becomes increasingly disturbing. The same is true for the frequency of bacterial resistance to antibiotics. The emergence and spread of resistance mechanisms acquired in the bacterial species now limit the indications of a number of first-line antibiotics, (Collee G *et al*, 2003).

The most common organism implicated in urinary tract infections (80–85%) is *E.coli*, (Nicolle LE, 2008) while *Staphylococcus saprophyticus* is the cause in 5–10%.Urinary tract infections (UTIs) are associated with high morbidity and long term complications like renal scarring, hypertension, and chronic renal failure. It also causes febrile illness, which often remain undiagnosed (Butler CC, et al; 2015.)

The main causative agent of urinary tract infection is *Escherichia coli*. Although urine contains a variety of fluids, salts, and waste products, it does not usually have bacteria in it (Barbosa-Cesnik C *et al* 2010). When bacteria get into the bladder or kidney and multiply in the urine, they may cause a UTI. Patterns of antibiotic resistance of these infections vary from year to year (Chawla R *et al* 2010). Monitoring of this resistance is needed to verify the validity protocols for first-line therapy and to suggest possible measures to control this evolution. This change in resistance is affirmed every year, raising fears of an inexorable trend towards inactivity of antibiotics. The multi-resistant strains, that is to say resistant to multiple antibiotics at once, have increased (Moustapha T, 2005). This motivated our study entitled: assessment of the antimicrobial susceptibility of *Escherichia coli* isolated from urine culture in bacteriology laboratory of the Bamenda Regional Hospital.

Escherichia coli are the leading cause of urinary tract, ear, wound and other infections in humans. Increasing rates of antimicrobial resistance among *E. coli* is a growing concern worldwide. Antimicrobial resistance in *E. coli* has been reported worldwide and increasing rates of resistance among *E. coli* is a growing concern in both developed and developing countries (Nicolle LE, 2008). A rise in bacterial resistance to antibiotics complicates treatment of infections. In general, up to 95 % of cases with severe symptoms are treated without bacteriological investigation (Raz *et al*, 2005). Due to the increase in occurrence of urinary *E. coli* infections and resistant bacterial strains over the last decade (Shadomy *et al* 1985), efforts have been made in the production of new antimicrobial agents which result in development in the clinical laboratory for selection and monitoring of antimicrobial chemotherapy. Efforts are now being made to standardise laboratory testing with these agents. Thus empirical antimicrobial therapy for urinary *E. coli* infections in the community where the Bamenda Regional Hospital is located is of major importance since it will go a long way to obtain medications of choice for this infection in the area. This research work of determining the antimicrobial susceptibility profile of *E. coli* will and has helped the society of this study population since it realises adequate treatment of choice for urinary E. coli infections in this study population of Bamenda. Also comparing present drug formulary of the ministry of public health in Cameroon with the researched profile will help deduce appropriate empirical therapy in this population of study thereby improving health care management.

2. LITERATURE REVIEW

2.1. Anti-microbial chemotherapy

Antimicrobial chemotherapy involves the use of chemicals in treatment of microbial infections. During the last 25 years, chemotherapeutic research was largely centred on antimicrobial substance of microbial origin called antibiotics. An antimicrobial is a chemical substance produced by microorganisms that can inhibit the growth of, or kill other microorganisms. Recently, chemical modification of molecules by biosynthesis has been a prominent new drug development method. Due to the increase in occurrence of fungal infections and resistant bacterial strains over the last decade, (Shadomy *et al*, 1985), efforts have been made in the production of new antimicrobial agents which result in development in the clinical laboratory for selection and monitoring of antimicrobial chemotherapy. Efforts are now being made to standardise laboratory testing with these agents (Chawla R *et al* 2010).

An ideal antimicrobial agent exhibits selective toxicity, thus a drug is harmful to a pathogenic microbe without harmful effects to the host. Selective toxicity may be a function of a specific receptor required for drug attachment and it depends on inhibition of biochemical events essential to microbes and not to the host (Fritsche T.R, 2005). Mechanism of action of these agents against microorganisms include: inhibition of cell wall synthesis, inhibition of cell membrane function, inhibition of protein synthesis and inhibition of nucleic acid synthesis by the microorganisms (Cooper R.A, 2003).

2.2. Escherichia coli

Escherichia coli (***E. coli***) is a Gram-negative, facultative anaerobic, rod-shaped bacterium that is commonly found in the lower intestine of warm-blooded organisms (endotherms). Most *E. coli* strains are harmless, but some serotypes can cause serious food poisoning in humans, and are occasionally responsible for product recalls due to food contamination. The harmless strains are part of the normal flora of the gut, and can benefit their hosts by producing vitamin K_2 (Bentley R et al; 1982), and by preventing the establishment of pathogenic bacteria within the intestine (Hudault *et al*, 2001).

E. coli and other facultative anaerobes constitute about 0.1% of gut microbiota, and fecal–oral transmission is the major route through which pathogenic strains of the bacterium cause disease. Cells are able to survive outside the body for a limited amount of time, which makes them potential indicator organisms to test environmental samples for fecal contamination (Feng P, et al; 2009).

The bacterium can also be grown easily and inexpensively in a laboratory setting, and has been intensively investigated for over 60 years.

2.3. Biology and biochemistry

E. coli is Gram-negative, facultative anaerobic and nonsporulating. Cells are typically rod-shaped, and are about 2.0 microns (μm) long and 0.5 μm in diameter, with a cell volume of 0.6–0.7 (μm)(Kubitschek HE, 1990). It can live on a wide variety of substrates. *E. coli* uses mixed-acid fermentation in anaerobic conditions, producing lactate, succinate, ethanol, acetate and carbon dioxide. Since many pathways in mixed-acid fermentation produce hydrogen gas, these pathways require the levels of hydrogen to be low, as is the case when *E. coli* lives together with hydrogenconsuming organisms, such as methanogens or sulphatereducing bacteria. Optimal growth of *E. coli* occurs at 37 °C (98.6 °F) but some laboratory strains can multiply at temperatures of up to 49 °C (120.2 °F) (Fotadar *et al* 2005) Growth can be driven by aerobic or anaerobic respiration,

using a large variety of redox pairs, including the oxidation of pyruvic acid, formic acid, hydrogen and amino acids, and the reduction of substrates such as oxygen, nitrate, fumarate, dimethyl sulfoxide and trimethylamine N-oxide. Strains that possess flagella are motile. The flagella have a peritrichous arrangement (Darnton NC *et al* 2007).

2.4. Parthenogenesis

Virulent strains of *E. coli* can cause gastroenteritis, urinary tract infections, and neonatal meningitis. In rare cases, virulent strains are also responsible for hemolytic-uremic syndrome, peritonitis, mastitis, septicemia and Gramnegative pneumonia (Todar, K, 2007). Uro-pathogenic *E. coli* (UPEC) is one of the main causes of urinary tract infections. Humans can be predisposed to *E. coli* in many ways. In particular for females, the direction of wiping of anus after defecation (wiping back to front) can lead to fecal contamination of the urogenital orifices. Anal sex can also introduce these bacteria into the male urethra, and in switching from anal to vaginal intercourse the male can also introduce UPEC to the female urogenital system (Evans Jr, 2007).

2.5. *E. coli* antimicrobial susceptibility

E. coli is one of the common cause of infections by gramnegative bacilli and the bacterial organism most often isolated from urine and blood cultures. It is a frequent cause of outpatient urinary tract infections in women worldwide, of hospitalization due to pyelonephritis and septicemia, and of nosocomial infections among hospitalized patients. Meningitis caused by *E. coli* in neonates is frequently fatal. Resistance to recommended first- and second-line agents, such as penicillins, cephalosporins, sulfa drugs (WHO, 2001.), and fluoroquinolones (Garau J et al; 1999). The choice of a specific antimicrobial agent or agents depends on local susceptibility patterns and on the part of the body that is infected

2.6. Activity and measurable qualities of antibacterial agent

Antibacterial agents possess varying specific activity and measurable qualities with respect to the different body tissues and their related pathogenic infections.

All beta-lactamase drugs as bacitracin, cephalospropins, cycloserine, penicillin and vancomycin are selective inhibitors of bacterial cell wall synthesis. The action mode here consists of binding of the drug to cell receptors as penicillin binding proteins and some transpeptidaton enzymes. Inhibition of the transpeptidase enzyme by penicillins and cephalosporins may be due to a structural similarity of these drugs and the transpeptidation reaction involves loss of a D-alanine from the pentapeptide. Insusceptibility to penicillin is in part determined by the organism's production of penicillin destroying enzymes (βlactamases). Bacitracin, vancomycin, ristocetin and novobiocin inhibit early steps in the biosynthesis of the peptidoglycan (Jawetz et al., 1991).

Antibiotics as chloramphenicol, tetracyclines, aminoglycosides, erythromycins and lincomycins act by inhibiting protein synthesis in bacteria. The concept of their mode of action is based on the subunit of each type of ribosome, their chemical composition and their functional specificities are sufficiently different to explain why antimicrobial drugs can inhibit protein synthesis in bacterial ribosomes without having a major effect on mammalian ribosomes (Jawetz *et al*., 1991). Antimicrobials as rifampin, quinolones, pyrimethanine, sulfonamides and trimethoprim act by inhibiting bacterial nucleic acid synthesis. Rifampin inhibits bacterial growth by binding strongly to the DNA-dependent RNA polymerase of bacteria. All quinolones and fluroquinolones inhibit microbial DNA-synthesis by blocking DNA gyrase.

Sulphonamide is involved in synthesis of folic acid (precursor to synthesis of nucleic acids) thus inhibits dihydropteroatesynthetase thereby preventing further growth of bacterial cell. Many bacteria that synthesize folic acid are consequently susceptible to action by sulfonamides (Jawetz *et al* 1991) . Trimethoprim inhibits dihydrofolic acid reductase 50,000 times (Jawetz *et al* 1991) more efficiently in bacteria than in mammalian cells. Pyrimethamine plus sulphonamide is the current treatment of choice in toxoplasmosis and some protozoal infections (Jawetz et al., 1991).

2.7. Urine culture

Urine cultures are performed to detect organisms that are the causative agents of urinary tract infections. Normally the urinary tract is sterile above the urethra. However, during non-invasive collection techniques urine is potentially contaminated with normal flora of the urethra and genitourinary tract. For this reason, urine cultures utilize a colony count (quantitation of growth) to aid in determining if dealing with contamination, colonization, or infection. Infections are associated with counts of 100,000 (10^5) or more organisms per ml of urine. However, low counts can be clinically significant in symptomatic patients. Selection of media and incubation requirements are based on the potential pathogens isolated. Common pathogens include but are not limited to:
Enterobacteriaceae, non-fermenting gram negative rods, Staphylococcus saprophyticus, Enterococcus, Group B Streptococcus and yeast (L. Ricci, 2008.)

2.8. Resistance to antimicrobial agents

Antibody mediated immunity (humoral) and cell-mediated immunity (cellular immunity), play little or no host immunity to fungal antigens as it is the reverse of this statement to protozoan antigens. This then demands the need for antimicrobial chemotherapy. Microbes can exhibit resistance to drugs in many ways:

- Microorganisms produce enzymes that destroy active drugs. For example, staphylococci pathogens produce β-lactamase that makes it to resist penicillin drugs.
- Microorganisms change their permeability to drugs. For example, streptococci have a natural permeability barrier to amino glycoside drugs.
- Microorganisms alter structural targets for some drugs. This is noticed in chromosomal resistance to amino glycosides by alteration of the specific protein in 30 "s" subunit of bacterial ribosome.
- Some microorganisms alter the metabolic partway that by passes the reaction inhibited by the drug.
- Some microbes alter enzymes that can still perform their metabolic functions. However, limitation of drug resistance may be minimised in the following ways:
- Maintenance of sufficiently high levels of drugs in tissues so as to inhibit both the original population and first step mutants.

- Simultaneously, administration of two drugs (synergism) that do not give cross resistance, with the other being able to delay emergence of mutant resistance to the drug. For example, Rifampin and isoniazid in the treatment of tuberculosis.
- Avoid exposure of microbe to particularly valuable drug by restricting its use, especially in hospitals and in animal feeds (G. M. Matar *et al* 2005)

3. MATERIALS AND METHODS

3.1. Laboratory procedures used in collecting data

The study put in place was prospective, descriptive and analytical since it involved empirical susceptibility testing. This study lasted from 15th of february 2019 to 11th of October 2019, carried out in the Bamenda Regional Hospital. The study population consisted of *E. coli* isolated from urine cultures during our study period. The Bamenda Regional Hospital is a public health institution located in the heart of Bamenda town. The town of Bamenda is the administrative capital and seat of government of the North West region. This town enjoys both rainy and dry seasons. It has good water supply and roads; educational and health facilities are also available.

3.2. Data collection by Administration of a questionnaire.

Detailed information relevant to the study was collected from each patient using a questionnaire (Appendix- 3). Such data included the age, province of origin, place of residence, religion, ethnicity, marital status, occupation and that of spouse, underlying clinical conditions, type of antibiotics often taken, whether they practice drug abuse, buy antibiotics from the street stores without prescription by a medical doctor, duration of use of particular antibiotics.

3.3. Sample Processing and Observation

Sample Population

This study was based on 50 E. coli isolates from the RHB Laboratory for antimicrobial sensitivity test within this period. There was no bias in the sample population every individual patient who came for consultation due to this infection and who willingly accepted to participate in the research was included. Their samples were collected and analyzed in the laboratory.

Sampling procedure

We systematically collected all *E. coli* isolated from urine cultures during our study period and having been the subject of antimicrobial susceptibility testing. Final data obtained from the analysis of patients samples on the number of patients who participated, then the drug of choice after susceptibility testing was identified.

Processing of specimens Urine Collection

Urine for a culture can be collected at any time. Due to the reason that urine can easily be contaminated with bacteria and cells from the surrounding skin during collection (particularly in women), it is important to first clean the genitalia. Women should spread the labia of the vagina and clean from front to back; men should wipe the tip of the penis. Start to urinate, let some urine fall into the toilet, then collect one to two ounces of urine in the sterile container provided, then void the rest into the toilet. This type of collection is called mid-stream clean catch urine.

Urine culture

It enables the isolation of bacteria and their numeration. On different Petri dishes, the identification number assigned to the sample matching is registered with. The homogenization of urine is carried gently stirring the pot of urine for a few seconds before seeding. The urine is then inoculated on CLED agar (Cystine-Lactose-ElectrolyteDeficient). The inoculated plates were then incubated at a temperature of 37 ° C for 18-24hours.

3.4. Direct examination

Macroscopic examination: We do macroscopic examinations on urine to determine its appearance (color, turbidity, odor, and abundance).

Microscopic examination

The technique used is that of the urinary sediment between slide and cover slip. This method is less reproducible. The pellet is placed between slide and cover slip and observed under the microscope objective 40 and this to appreciate the cellular components of urine (erythrocytes, leukocytes, cells epithelial cells, Trichomonas, sperm, yeast, eggs bilharzia, crystals, cylinders ...).The samples with a high white blood cell count (a few, quite a few and many leukocytes) are subject to the Gram stain.

Sample collection

Cultural response on CLED Agar at the appropriate atmosphere and temperature and examined for growth at 18 – 24 hours incubation. Bromothymol blue indicator in the agar changes to yellow due to acidification of the medium due to lactose fermentation by *E.coli* growth.

3.5. In vitro Antimicrobial susceptibility testing (Disk diffusion method).

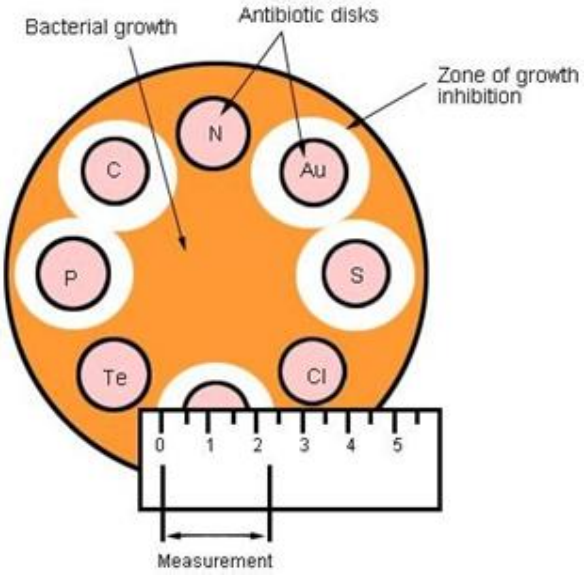
The antimicrobial susceptibility testing was carried out, using the disk diffusion technique of Bauer et. al., (1966), modified and standardized by the National Community for Clinical Laboratory Standard (Lalitha, 2005).

The antibacterial susceptibility testing on *E.coli* was carried out on Mueller Hinton agar using discs (ABTEK- BIOLOGICAL Ltd LIVERPOOL, UK) with the following drug contents: Amoxicilline(25µg), Amoxicilline +A. clavulanique (20µg + 10µg), Céfotaxime(30µg), Céftriaxone(30µg), Peniccilin(10µg), Ciprofloxacine(5µg), Doxicycline(5µg), Tetracyclin (30µg), Erythromycin(15µg), Penicillin(10µg) The antibacterial testing was carried out on fresh isolates of *E coli* on Mueller Hinton agar at 37°C for 24 hours.

3.6. Standardisation of innoculum.

The inocula were prepared from pure isolates grown on CLED agar at 35°C for 24 hours. Five pure colonies of each strain on the 24 hours old cultural plates were randomly selected, touched and inoculated into 5 ml of sterile 0.85% normal saline in bijoux bottles. The turbidity of the cell suspension was adjusted to match that of a 0.5Mc Farland Barium Sulphate standard containing approximately $1x\ 10^6$ cells/ml of the inoculum. A sterile cotton swab was dipped into the standardised respective microbial suspension; drained off and then used to inoculate the dry surface of the Mueller Hinton agar plate. The antibacterial discs were each aseptically placed on the inoculated plate using a sterile forcep and then incubated at 37°C for 24 hours

3.7. Measurement of the diameter of growth inhibition.



Presentation and discussion of results

The diameter of the growth inhibition surrounding each of the microbial agents was measured in millimetres (mm) using a ruler. The results were interpreted as sensitive, intermediate and/or resistant according to the National Community for Clinical Laboratory Standard (appendix-1).

Data Analysis

Data collected was presented on tables, Pi-chart and graphs. From the data that was collected. The prevalence of the disease was calculated using following formula.

$$\text{Prevalence} = \frac{\text{Total number of old and new cases with E.coli infection}}{\text{Total number of patients in the hospital.}} \times 100$$

4. DATA PRESENTATION AND OR RESULTS

4.1. General presentation of data

A total of 75 culture respondents were isolated using the CLED agar, out of these, 50 (66.7%) were *E. coli* positive against 25(33.3%) others bacteria . In the antibiotic susceptibility testing, there was 81% susceptibility to Ciprofloxacine, Cefotaxime (68%), Ceftriaxone (56%), Gentamycine (24%), Erythromycin (6%), Doxycycline (12%), Penicillin (18%), Tetracyclin (0%) (Table 1: below).

Table 1: Distribution of 50 *Escherichia coli* according to antibiotic susceptibility

	S	I	R	Total
Amoxi +A.clavunanique	13(26%)	19(38%)	6(36%)	50(100%)
Amoxicilline	9(18%)	16(32%)	25(50 %)	50(100%)
Erythromycin	3(6%)	13(26%)	34(68%)	50(100%)
Céfotaxime	34(68%)	13(26%)	3(6%)	50(100%)
Céftriaxone	28(56%)	9(18%)	13(26%)	50(100%)
Doxicycline	6(12 %)	3(6 %)	41(81 %)	50(100%)
Gentamicine	12(24 %)	22(44 %)	16(32 %)	50(100%)
Ciprofloxacine	41(81 %)	6(12 %)	3(6 %)	50(100%)
Tetracyclin	0(0 %)	19 (38 %)	31(62%)	50(100%)
Penicillin	9(18 %)	16(32 %)	25 (50 %)	50(100%)

S = susceptible I = moderate susceptibility R = resistant

DISCUSSION

Identification of E. coli was done on the basis of their morphological and biochemical characteristics. The sensitivity study was done by disk diffusion technique on Mueller-Hinton. The interpretation susceptible, intermediate and resistant was made in accordance according to the National Community for Clinical Laboratory Standard (appendix1). In the total bacterial population, we note a predominance of Escherichia coli (66.6%). In this study, the result of the anti-bacterial susceptibility showed that ciprofloxacin 81%, was highly effective and to a lesser extent to the third generation cephalosporin (Céftriaxone 56% and Céfotaxime 68%). High resistance was recorder for amoxicillin (50%), penicillin(50%), Amoxicilline +A. clavulanique(36%), tetracyclin(62%), erythromycin(68%), doxicycline(81%), gentamycin(32%). Similar studies conducted in Ethiopia and Nigeria reported comparable susceptibility rates with high sensitivity to ciprofloxacin and

gentamicin and norfloxacin (M *Kibret and B Abera, 2011). As similar to this study, high resistance rates of *E. coli* was observed in a study at Ibadan in Nigeria and it showed amoxycillin (100%), cotrimozazole (92.85%) and tetracycline (100%), (Joseph OmololuAso et al; 2017). Third-generation cephalosporin such as ceftriaxone has been used to treat Gram-negative bacterial infections of various body sites and this might be as a result of Extended Spectrum Beta-Lactamases (ESBL) in the strains.

Traditional medicine and drug abuse may greatly contribute to drug resistance development in this environment.
Traditional medicine and drug abuse may greatly contribute to drug resistance development in this environment.

5. CONCLUSION AND RECOMMENDATIONS

5.1. CONCLUSION

At the end of our prospective study, urine was sampled at the Laboratory of Medical microbiology Bamenda Regional Hospital and the study was focused on 50 *E. coli* isolates samples. Ciprofloxacin was highly effective and to a lesser extent to the third generation cephalosporin (Céftriaxone and Céfotaxime). Meanwhile amoxicillin, penicillin, Amoxicilline +A. clavulanique, tetracyclin, erythromycin, doxycycline and gentamycin were not effectively against *E coli* urine isolates in this study area.. The monitoring of these developments is necessary to verify the validity of protocols to the first-line treatment. The irregularity of the sensitivity of the bacteria to antibiotics coupled with the involvement of this species in most infections imposes a periodic reassessment of their sensitivity to antibiotics.

5.2. RECOMMENDATIONS

At the end of this study, we make the following recommendations: ➢Laboratory technicians:

- Always follow good laboratory practice;
- Be more supportive in the work;
- Repeat as often as possible similar studies to monitor levels of bacterial resistance.

Prescribers:

- Avoid systematic prescription of a type or family of ATB;
- Ask the possible susceptibility testing before considering antibiotic therapy;
- Properly equip the laboratory feature is the imperative to provide a catalog API idenfication can enable faster and better identification;
- Provide laboratory reagents and consumables sufficient quality and quantity to enable it to fulfill its role of regional reference laboratory;
- Building an archive room of the laboratory;
- The extension of the results to all medical and paramedical personnel.

Ministry of Health:

- Implement adequate means of epidemiological surveillance antibiotic sensitivity of bacterial strains isolated in laboratories;
- Finance study to study the phenotypes of resistance to study the evolution of mechanisms of resistance of bacteria isolated.

Populations

- Avoid self medication.

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APPENDIX-1: **Appendix-1Interpretative break point for *E.coli* strains.**

Antimicrobials	Disc potency	Zone diameter of growth inhibition (mm).		
		Resistant (<)	Intermediate	Sensitive (>)
Amikacin	10µg	18	-	24
Ampicillin	10µg	24	-	35
Bactracin	10 Unit	17	-	22
Cephalothin	30µg	25	-	37
Chloramphenicol	10µg	17	-	18
Clindamycin	2µg	23	-	29
Erythromycin	15µg	23	-	30
Gentamycin	10µg	19	-	27
Kanamycin	30µg	19	-	26
Methicillin	5µg	17	-	22
Neomycin	30µg	18	-	26
Augmentin	10µg	18	-	24
Penicillin	10µg	26	-	37
Polymycin B	300 Units	7	-	13
Streptomycin	10µg	14	-	22
Sulfamethoxazole-trimethopim	25µg	24	-	32
Tetracycline	30µg	19	-	22
Tobramycin	10µg	19	-	29
Ciprofloxacin	1µg	17	-	19
Vancomycin	30µg	15	-	19