Antibiogram and Susceptibility of *Klebsiella* Spp Isolated from Different Clinical Specimens in Health Care Centers in Etsako West Local Government Area of Edo State, Nigeria

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Abstract

Arising multidrug resistance to available antibiotics is a problem of deep scientific and research concern. One organism with increasing antibiotics resistance is *Klebsiella* spp. that had cause several outbreaks in different locations. This study was undertaken to investigate the antibiogram and susceptibility of *Klebsiella* spp. isolated from clinical samples in health care centers. The study was conducted in Auchi Township in Etsako West Local Government Area, Edo State, Nigeria. Following standard procedures samples were collected and *Klebsiella* spp. identified using standard microbiology techniques. Antibiotic susceptibility testing was carried out using standard methods. 109 samples were collected and comprised urine (44), wound (20), high vagina swabs (17), sputum (9), semen (6), stool (5), endo-cervical swab (2), throat (1) and ear (1). 10 (9.17%) samples were positive to *Klebsiella* organism with urine consisting 50.0% and 10.0% each from sputum, stool, wound, ear and high vaginal swabs. *Klesiella* isolates were completely resistance to penicillin, Amoxiclav and Chloramphenic. Amoxicillin, Floxapen, Metronidazole, Ampicillin, Cotrimoxazole, Nalidixic acid, Streptomycin, Tetracycline, Cephalexin, Gentamic, Tobramycin, Nitrofurantoin, Ciprofloxacine and Cefuroxime have less than 50% potency. On the other hand, Ofloxacine, Cefotaxime, Ampisulbactam and Perflacine were 50% and above susceptible. *Klesiella* spp. isolated from stool sample was more multi-drug resistance (87.75%) followed by those from wound (77.55%), sputum (73.47%), high vaginal swab (69.39%), urine (65.30%) and lastly ear sample (48.98). Based on our findings, *Klesiella* spp. in the study area is multidrug-resistance thus suggesting antibiotic analysis prior to treatment.

Keywords: *Klebsiella* Spp; Antibiogram typing; Clinical specimen; Etsako

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Introduction

*Klebsiella* species is a Gram negative opportunistic pathogen and well known to clinicians as a cause of community acquired bacterial pneumonia, occurring particularly in chronic alcoholics, urinary tract infections, wound infections, blood infections and infections in the intensive care unit [1, 2]. The genus *Klebsiella* comprises of five species, *Klebsiella pneumoniae*, *Klebsiella oxytoca*, *Klebsiella planticola*, *Klebsiella terrigena* and *Klebsiella ornithinolytica* [3]. Of all five species, *Klebsiella pneumoniae* is the most common hospital-acquired pathogen causing lung infections, wound infections, meningitis, abscesses, urinary tract infections and diarrhea [4] and has been reported to cause outbreak of sepsis and death of newborns in the intensive care unit in Brazil [5], United Kingdom [6] and France [7]. It was therefore identified as an important common pathogen causing nosocomial pneumonia (7 to 14% of all cases), septicemia (4 to 15% of all cases), wound infections (2 to 4% of all cases) and neonatal septicemia (3 to 30% of all cases) [8]. In addition, it is said to be a normal flora of the intestine and are found in respiratory tract of humans and animals [2]. While cockroaches were suggested to play the role of vectors of this hospital-acquired pathogen, they have also been found in aquatic environments and industrial waste waters [9], plant products and fresh vegetables [10].

As an opportunistic pathogen, *Klebsiella* spp. primarily attacked immune-compromised individuals, who are hospitalized and suffer from severe underlying diseases such as diabetes mellitus or chronic pulmonary obstruction, chronic cardiac, renal and neoplastic disease [2, 11-12]. Worrisome, reports suggest that *Klebsiella* is emerging worldwide and has become a serious threat to human health by causing endemic and epidemic infections [13]. Although progress has been made in approaches to infection treatments and antibiotics production, bacteria have developed resistant against available antibiotics and have made the treatment of infectious diseases even more difficult [14].

For example, beta-lactam antibiotics or alternatively with aminoglycosides or fluoroquinolones are often the drugs of choice for infections caused by *Klebsiella* spp. however, resistant of this organism to these antibiotics have been reported [15-17]. Development of antibiotic resistance in *Klebsiella* spp. and the production of multidrug-resistant strains have cause infections by these strains to become challenging [18]. In 1980, the resistant of *Klebsiella* strains to the cephalosporins such as oxyimino beta-lactams (ceftriaxone, ceftazidime, cefotaxime) was reported and recorded for the first time and since then *Klebsiella* bacteria were ever more resistant to antibiotics [19].

In fact, multidrug resistant *Klebsiella* pneumonia has been reported [20] and was suggested to be due to plasmid encoding extended-spectrum β-lactamases (ESBLs). At the level of a wider geographic scale, the incidence of extended-spectrum β-lactamases (ESBL) producing organisms was reported to be difficult to resolve due to difficulty in detecting ESBL production and inconsistencies in reporting [21]. Recent several studies have documented significant increase in the incidents of ESBL-related infections and it’s observed throughout the globe [22-28]. Despite this reports of multiple antibiotics resistance and variation between geographical locations, few studies have been conducted in Nigeria with a few in Edo State and non in Etsako West Local Government Area of Edo State. This study therefore was embarked on to investigate the
antibiogram and susceptibility of *Klebsiella* spp. isolated from different clinical specimen in a Medical Center, a laboratory and a general hospital in Etsako West Local Government Area of Edo State.

**Materials and Methods**

**Study area:** This study was carried out in Auchi township and environ (7º04'N, 6º16'E) in Edo State, Nigeria between 15th of January to 15th of April 2015. The area is the head-quarters of the Etsako West Local Government Area and a host to a tertiary institution (Auchi Polytechnic, Auchi), several secondary and primary school, several clinic, laboratories and government owned health care facilities.

**Sampling:** A total of 109 clinical specimens which includes urine, blood, stools and swabs (wound, throat, and ear swabs) were collected from randomly selected clinics consisting of a private clinic (Fate Medical Center), a government owned health facility (General hospital, Auchi) and a medical laboratory (Medi-view laboratory) in Auchi Township. Specimens were transported to the diagnostic laboratory of Fate Foundation Hospital and stored at 4ºC refrigerator before culturing, isolation and provisional identification. Analysis was done within 24hrs of collection. Isolates were placed on Nutrient agar slopes and stored in the refrigerator. Periodically, isolates were transferred to the microbiology laboratory at Ambrose Alli University Ekpoma for confirmatory identification.

**Identification of Isolates:** Isolates were cultured on MacConkey agar and nutrient agar under aseptic conditions [29-31]. The MacConkey agar and Nutrient agar plates were incubated at 37ºC. After overnight incubation, growths of suspected organisms were identified by their colonial morphology and biochemical characteristics [32]. These tests include the following:

1. **Gram Stain:** Discrete colonies of the test organisms were obtained aseptically with sterile wire loop and emulsified in drop of normal saline on a clean sterile glass slide. The smear was air dried and then heat fixed by passing it slightly through a Bunsen burner flame. Then the fixed smear was covered with crystal violet stain on a staining rack for 30 seconds, and then washed off with distilled water. The smear was then flooded with luguol’s iodine for one minute, and then rinsed with distilled water. The smear was decolourized with acetone for 10 seconds and was immediately washed off with distilled water. The smear was counter stained with neutral red for one minute, washed off and then allow to air dry. A drop of immersion oil was placed on the stain potion of the slide and it was viewed under x100 objective lens. All Gram negative organisms were pinkish or pale to dark red in colour, which is as a result of the secondary dye while gram positive organisms were usually purple in colour which was the colour of the primary dye.

2. **Motility Testing:** A molded plasticine ring was set on a clean slide. A loopful of an overnight broth culture of the test organism was taken unto a cover-slip, and the slide was inverted on the cover-slip making sure that the culture drop was within the plasticine ring but without touching the ring. This was inverted and examined under the x10 and x40 objectives.

3. **Catalase Test:** Catalase test was carried out by placing a drop of hydrogen peroxide on a clean grease free glass slide. A colony of the test organism was taken with a sterile platinum wire loop,
and emulsified in the hydrogen peroxide on the slide. The production of gas bubbles which indicated positive result was then observed.

4. Oxidase Test: A piece of filter paper was placed on a clean Petri-dish and two drops of freshly prepared oxidase reagent was added. Then a colony of the test organism was smeared on the oxidase reagent soaked filter paper. Development of a deep purple colour within 10 seconds indicated positive result.

5. Urease Test: This test the ability of certain organisms to produce the enzyme urease which decomposes urea into alkaline, ammonia and carbon (iv) oxide. The test organism was inoculated heavily in bijou bottle containing 3ml of sterile Christensen’s modified urea broth. It was incubated at 37°C for 12 hours. A pink colour in the medium indicated a positive result.

6. Coagulase Test: This test is used to differentiate Staphylococcus aureus which produces the enzyme coagulase, from S. epidermidis and S. saprophyticus which do not produce coagulase. Coagulase causes plasma to clot by converting fibrinogen to fibrin. Two types of coagulase are produced by most strains of S. aureus. Free coagulase converts fibrinogen to fibrin by activating a coagulase reacting factor present in plasma and this is detected by the appearance of a fibrin clot in the tube test while bound coagulase (clumping factor) converts fibrinogen directly to fibrin without requiring a coagulase –reacting factor. It can be detected by the clumping of bacterial cells in the rapid slide test. A slide coagulase test is run with a control to rule out auto agglutination. Two drops of physiological saline are put onto a slide labeled with test (T) and control (C), the two saline drops are emulsified with the test organism using a wire loop. A drop of plasma is placed on the inoculated saline drop corresponding to test, and mixed well, then the slide is rocked gently for about ten seconds and observes for clumping. No clumping is an indication of a negative result. If slide coagulase is negative this is followed by a tube test for further confirmation. The tube test uses rabbit plasma that has been inoculated with Staphylococcal colony (i.e. Gram positive cocci which are catalase positive). The tube is incubated at 37°C for 1 hour. If negative, then incubation is continued up to 18 hours. If positive, the plasma will coagulate.

7. Indole Test: This test is used to demonstrate the ability of certain bacteria to decompose the amino acid tryptophan to indole which accumulates in the medium; the organism is inoculated into peptone water and incubated overnight at 37°C. The presence or formation of indole is tested by adding a few drops of kovac’s reagent (0.2-0.3). The test tube is well shaken and allowed to stand, the formation of a dark red colouration indicates a positive result and no colour change indicates a negative result.

8. Citrate Test: Slant of medium containing citrate and ammonium salt was prepared in bijoux bottles and stored at 4°C in the refrigerator, with the use of a sterile wire; the slant was streaked with a saline suspension of the test organism and then stabbed into the medium followed by incubation at 35°C for 48 hours. A bright colour indicated positive result.

9. Indole Test: Bijou bottles containing 3ml of sterile peptone water was inoculated with test organism, and then incubated at 37°C for 48 hours. Thereafter, 0.5ml Kovac’s reagent was added and shaked gently. Positive result was indicated by the formation of red colour in the surface layer within 10 minutes.
Antibiotic susceptibility testing: Prepared antibiotic discs were used as follows; A 1/100 dilution of an overnight nutrient broth culture of each test organism was poured on the surface of nutrient agar plate that was notched round. The excess broth was discarded into a disinfectant jar. The plates were left to stand on the bench top for 30 minutes. After 30 minutes, the antibiotic discs were placed on the surface of the inoculated agar medium. The plates were incubated at 37°C for 24 hours after which the zones of inhibition were observed and recorded. Zones of up to 13 mm diameter or over were recorded as sensitive while those less than 13 mm were regarded as resistance [29].

Preparation of antibiotic susceptibility discs: In cases where the commercially prepared antibiotic discs were not available, the discs were personally prepared as follows: Whatman No.1 filter paper discs with diameter of 6 mm were punched out with a paper puncher. One hundred of the discs were counted into bijoux bottles. The bottles and the discs were sterilized in a hot air oven at 140°C for 2 hrs. The bottle and its contents were allowed to cool to room temperature. The discs were then aseptically inoculated with 1 ml of the pre-prepared antibiotic solution. The bottles’ caps were partly screwed down and allowed to dry overnight in a 37°C incubator [29].

Antibiogram typing: This was done by dividing the 21 antibiotics into seven different groups of 3 antibiotics each according to their mode of action, similarities and usage [33]; antibiotics were arranged in ascending order of their molecular weight or their generations in each group of 3 antibiotics viz; Group 1: Ampicillin, Amoxicillin and Cloxacillin; These are bacteria cell wall synthesis inhibitors. They impair cell wall synthesis by preventing cross-binding of the peptidoglycan polymers necessary for cell wall formation and by binding the penicillin-binding proteins (PBPs) (carboxypeptidases, endopeptidases, and transpeptidases) that participate in cell wall synthesis.

Group 2: Flucloxacillin, Amoxyclav and Ampisulbactam; These are broad-spectrum penicillin with a fluorinated side chain in the first and attached clavulanic acid in the other two

Group 3: Genticin, Tobramycin and Streptomycin; These are Aminoglycocides- broad spectrum, protein synthesis inhibitors, active against aerobes only.

Group 4: Chloramphenicol, Tetracycline and Cotrimoxazol; Seprtin is a folic acid inhibitor while the other two are protein synthesis inhibitors. Here, they represent the most commonly used (abused) antimicrobials in the community.

Group 5: Metronidazole, Nitrofurantoin and Nalidixic acid; Metronidazole is anti-protozoan but is also active against anaerobic and facultatively anaerobic bacteria; Nitrofurantoin is active against Gram-positive and Gram-negative bacteria and is mostly used as urinary antiseptic while antimicrobial activity of early quinolones (the first generation quinolones) such as Nalidixic acid, are excellent against aerobic Gram-negative bacteria.

Group 6: Ofloxacin, Ciproxin and Peflacine; Quinolones act by inhibiting the action of topoisomerasases II (DNA gyrase) and topoisomerase IV. For Gram-negative bacteria the prime target of quinolones is the
DNA gyrase, whereas in the Gram-positives it is the topoisomerase IV. These three quinolones act by inhibiting the DNA gyrase.

Group 7: Cephalexine, Cefotaxime and Cefuroxime; Cephalexin is an oral penicillin substitute that is active mainly against Gram-positive organisms while the latter two are broad spectrum and highly active against Gram-positive and Gram-negative organisms. These drugs are highly expensive, and they are usually not abused.

**Ajumali's method of pneumonic coding** [34]: This is an adaptation of Ajumali’s method of pneumonic coding [33,34]. Sensitive result was scored as (+) while resistance was recorded as (-). The three antibiotics in each group were given numerical values of 1, 2, and 4. A perfect sensitivity to the three antibiotics will give a summation of 1+2 +4 = 7. On the other hand, complete resistance to the three antibiotics will give a summation of 0+0 +0 =0. Other values are obtained by adding up these numerical values; in which case an isolate can receive a score of 0-7 in each triplet segment. Combining the seven-triplet segments together then gives a seven-digit numerical value as the antibiogram type.

**Materials/Instrumentation and Techniques:** The equipment used in this study include the following; hot air oven, incubator, Aluminium foil paper, Petri dishes, measuring cylinder, Bijou bottles, Cotton wool, test tubes, sterile hand gloves, Masking tape, glass slides, Nutrient agar, Mueller Hinton agar, Normal physiological saline, acridine orange/sodium dodecyl sulphate. Media were prepared according to the manufacturer’s instruction. Cultural characteristics were observed using standard microbiological techniques. Pure cultures were isolated, Gram stained, followed by biochemical tests to identify the isolates. All glass wares were washed with detergent, rinsed in distilled water and sterilized at 100°C in the hot air oven for 1 hour before use while wire loops were sterilized by passing them through a Bunsen burner flame until it was red hot before use.

**Results and Discussion**

Analysis of the results showed that the collected clinical specimens comprised of 55 females (50.5%) and 54 males (49.4%) samples. The number of clinical specimens obtained were 44 urine samples, 20 wound samples, 17 high vagina swabs, 3 urethral swabs, 9 sputum samples, 6 semen samples, 5 stool samples, 2 endo-cervical swab and 1 each of throat and ear swabs (see Table 1). Of the 109 clinical samples examined, 10 were associated with *Klebsiella* organisms. This gives a prevalence rate of 9.17%. This is high compared to the 5.11% (100 *Klebsiella* isolates from 1957 clinical samples) reported for a rural teaching hospital in India [35] or the 2.4% (24 *Klebsiella* isolates from 1000 clinical samples) reported for Federal Teaching Hospital Abakaliki, Ebonyi State [36] or the 2.99 reported for the National hospital Abuja, Central Nigeria [37]. The observed prevalence of *Klebsiella* in our study is low compared to the 17.3% (*Klebsiella pneumoniae*) reported for a tertiary health care facility in Yola, Northern Nigeria [38]. 38.5% (150 *Klebsiella pneumoniae* isolates from 390 clinical samples) reported for University of Nigeria Teaching Hospital, Ituku Ozalla in Enugu capital city in South-Eastern Nigeria [39]. Our finding therefore suggests that *Klebsiella* spp is prevalent in the clinical samples and by implication in clinics within Auchi Township area of Estako West Local Government Area of Edo State.
Specifically, of the 10 *Klebsiella* organisms isolated, 5 (50.0% of K. spp. isolates) were from urine sample, 1 each (10.0% of K. spp. isolates) was from sputum, stool, ear swab, wound and high vaginal swab samples (see Table 1). The observed higher representation of *Klebsiella* infestation in urine sample (50.0%) in our study is similar to the finding by Ali Abdel Rahim and Ali Mohamed [40], where isolation rate of *Klebsiella* spp was said to be more from pus, followed by urine, sputum and catheter tip. Romanus and Egwu [39] have also reported similar distribution of *Klebsiella* infestation. Our finding therefore indicates that *Klebsiella* infestation is more likely related to urinary tract infections.

### Table 1 Distribution of *Klebsiella* bacteria isolates from specimen according to clinical samples

<table>
<thead>
<tr>
<th>Specimens</th>
<th>Urine</th>
<th>Sputum</th>
<th>Semen</th>
<th>Stool</th>
<th>ECS</th>
<th>Throat swab</th>
<th>Ear swab</th>
<th>Wound</th>
<th>HVS</th>
<th>Urethral swab</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sample collected</td>
<td>44</td>
<td>9</td>
<td>6</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>20</td>
<td>17</td>
<td>3</td>
<td>109</td>
</tr>
<tr>
<td><em>Klebsiella</em> spp</td>
<td>5</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>% in specimen</td>
<td>11.36</td>
<td>11.11</td>
<td>0.00</td>
<td>20.00</td>
<td>0.00</td>
<td>0.00</td>
<td>50.00</td>
<td>5.00</td>
<td>5.88</td>
<td>0.0</td>
<td>9.17%</td>
</tr>
</tbody>
</table>

**KEY:** HVS = High vagina, ECS = Endo-cervical swab

![Figure 1](image-url)  
**Figure 1** Comparative susceptibility and resistibility of *Klebsiella* spp isolates to antibiotics (Key %R = percentage resistivity, %S = percentage sensitivity).

Antibiogram typing of the isolated *Klebsiella* spp. (Table 2) showed that ear swab isolate was more sensitive (51.02%) to antibiotics. This was followed by *Klebsiella* spp isolated from urine samples (34.70%), high vaginal swab (30.61%), sputum (26.53%), wound (22.45%) and lastly stool sample (12.25%) (see Table 2). This finding disagrees with the report by Romanus and Egwu [39], who
observed that *Klebsiella* pneumoniae from wound samples were the most susceptible strains followed by HVS, sputum and urine. In our study, *Klebsiella* spp. isolated from stool sample was more multi-drug resistance followed by those from wound sample, sputum, high vaginal swab, urine and lastly ear sample (see table 3).

Table 2 Antiogram types and antibiotics sensitivity of the clinical *Klebsiella* spp. isolates

| Conc. | Penicillin | Ampicillin | Amoxicillin | Floxapen | Amoxilbacum | Gentamic | Streptomycin | Tetracycline | Chloramphenicol | Cotrimoxazole | Nitrofuranto0n | Metronidazole | Nalidixic acid | Ofloxacine | Ciprofloxacine | Perflacine | Cefalexin | Cefotaxine | Cefuroxime | Types | Sensitivity | % sensitivity |
|-------|------------|------------|-------------|-----------|-------------|----------|-------------|--------------|----------------|--------------|----------------|---------------|----------------|----------------|-------------|-------------|-----------|-----------|-----------|-----------|--------|-----------|----------------|
| S     | 1 2 4 1 2 4 1 2 4 1 2 4 1 2 4 1 2 4 1 2 4 | - + - - - + + - + - + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + + ...
Ciproxin and Peflacine) presented the highest antibiotic potency to *Klebsiella* spp. (see table 2). The high resistance of *Klebsiella* spp. to amoxicillin antibiotic (R=90.0%) in this study is similar to the study by Dallal et al. [41] and Langarizadeh et al. [42] who reported 97% and 98.61% respectively.

**Table 3** Comparative resistivity pattern of *Klebsiella* spp. isolated from different clinical specimen to antibiotics

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>%R Urine</th>
<th>%R Sputum</th>
<th>%R Stool</th>
<th>%R Ear swab</th>
<th>%R Wound</th>
<th>%R HVS</th>
<th>% S</th>
<th>% R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>60.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>20.0</td>
<td>80.0</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
<td>100.0</td>
<td>10.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Floxapen</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
<td>10.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Amoxiclav</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Ampisulbactam</td>
<td>40.0</td>
<td>100.0</td>
<td>0.0</td>
<td>0.0</td>
<td>100.0</td>
<td>100.0</td>
<td>60.0</td>
<td>40.0</td>
</tr>
<tr>
<td>Genticin</td>
<td>60.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
<td>100.0</td>
<td>40.0</td>
<td>60.0</td>
</tr>
<tr>
<td>Streptomycin</td>
<td>60.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
<td>100.0</td>
<td>30.0</td>
<td>70.0</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>60.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
<td>0.0</td>
<td>40.0</td>
<td>60.0</td>
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<tr>
<td>Tetracycline</td>
<td>80.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>0.0</td>
<td>30.0</td>
<td>70.0</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Cotrimoxazole</td>
<td>80.0</td>
<td>0.0</td>
<td>100.0</td>
<td>100.0</td>
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<tr>
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<tr>
<td>Cefotaxime</td>
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<tr>
<td>Cefuroxime</td>
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<tr>
<td><strong>Total Resistance</strong></td>
<td><strong>68.10</strong></td>
<td><strong>80.95</strong></td>
<td><strong>90.48</strong></td>
<td><strong>52.38</strong></td>
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<td><strong>66.67</strong></td>
<td><strong>29.05</strong></td>
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The antibiotics susceptibility observed in our study were in variants compared to the findings by Romanus and Egwu [39] who reported higher susceptibility profiles as follows; imipenem (100%), amikacin (100%), ceftazidime (98%), cefoxitin (99.4%), cefotaxime (96.7%), amoxicillin/clavulanic acid (96%), ciprofloxacin (96%), tobramycin (93.3%), kanamycin (90%), cefuroxime (86.7%), gentamicin (76%), sulphonmethoxazole/trimethoprim (22%), chloramphenicol (15.4%) and ampicillin (5%). On the other hand, Abubakar (2009) reported lower susceptibility compared to Romanus and Egwu [39] but higher than what was observed in our study. Specifically, Abubakar [38] reported susceptibility profile of Klebsiella pneumoniae to be 65.4% (augmentin), 58.5% (ofloxacin), 55.0% (nalidixic acid), 52.7% (erythromycin) 51.7% (nitrofurantoin), 50.5% (chloramphenicol), 50.0% (colistin), 49.0% (streptomycin), 38.8% (pfofloxacin and gentamycin respectively), and 42.3% (ampicillin).

The variations in antibiotics susceptibility between our presence study and others studies indicate differences in multiple antibiotic resistance (MAR) of Klebsiella spp. In fact, nosocomial isolates are now reported to be now frequently resistant to numerous antibiotics. This was said to be due to acquisition of multidrug resistance (MDR) plasmids and Klebsiella pneumoniai has been identified as one of the most common organisms to carry plasmid encoding extended-spectrum β-lactamases (ESBLs), and such strains are isolated with increasing frequency [43]. Klebsiella oxytoca has also been reported to be resistant to multiple antibiotics due to the chromosomal beta-lactamase over expression that leads to a characteristic antibiogram with high resistance to many available antibiotics [43, 44]. The acquisition of multidrug resistance (MDR) plasmids and chromosomal beta-lactamase over expression may be the reason for the low susceptibility and high resistivity reported in our study. This assertion is based on the obvious indiscriminate utilization of antibiotics by our population due to poverty and expensive delivery of health care services. The variations in antibiotics susceptibility potency between our study and previous studies, point toward the need for monitoring of antibiotic treatment and multiple antibiotic resistances.

**Conclusion**

Judging by the findings of this study, Klebsiella spp. possesses high antibiotic resistance to available antibiotics. The 100% resistance of Klebsiella spp. to some antibiotics in this study indicates serious problem with antibiotic treatments. In addition, the low sensitivity of many antibiotics observed in this study indicates Klebsiella organisms to be gaining renewed multidrug resistance among the available antibiotics. Thus, suggesting a threat to patients’ effective management, especially in developing countries where antibiotic susceptibility is not conducted routinely. It is therefore our recommendation that findings on antibiogram typing, antibiotic susceptibility and resistivity be communicated to clinicians and pharmacists for effective patient management.

**Acknowledgement**

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