

Prevalence and Antibiotic Susceptibility Pattern of *Pseudomonas aeruginosa* and *Escherichia coli* O157:H7 Isolated from two Hospital Environments in Zaria, Nigeria

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Authors' contributions

This work was carried out in collaboration between both authors. Author CH designed the study, performed the statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. Authors HLY and CH managed the analyses of the study. Author CH managed the literature searches. Both authors read and approved the final manuscript.

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ABSTRACT

Aims: The aim of this study is to determine the prevalence and antibacterial susceptibility of *Pseudomonas aeruginosa* and *Escherichia coli* and to screen for *Escherichia coli* O157:H7.

Study Design: Samples for the studies were collected from surfaces in the hospitals. A total number of 310 samples were collected from the two hospitals; 155 samples were collected.

Place and Duration Studies: Department of Microbiology, Ahmadu Bello University, Zaria, Nigeria. The work was carried out between May 2012 and March 2013.

Methodology: The pathogens were isolated using differential Biochemical tests both conventional and kits were carried out for the identification of the *P. aeruginosa* and *E. coli*. The isolates of *Escherichia coli* were screen for *E. coli* O157:H7 using serology test. The antimicrobial susceptibility pattern was determined using Kirby-Bauer-CLSI modified single disc diffusion

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technique.

Results: The results from Major Ibrahim B. Abdullahi memorial hospital showed that 16 (69.6%) and 7 (30.4%) were *P. aeruginosa*, and *E. coli* respectively. And from St. Luke Anglican hospital 13 (61.9%), *P. aeruginosa* and 8 (38.1%) *E. coli* were isolated. There was zero (0) prevalence of *Escherichia coli* O157:H7 in the two hospitals selected for the study. *Pseudomonas aeruginosa* was 100% susceptible to ceftazidime and imipenem, *E. coli* was also 100% susceptible to gentamicin, cefoxitin and ceftazidime. *P. aeruginosa* and *E. coli* were more resistant to tetracycline. The multidrug resistant of the isolates from Major Ibrahim B. Abdullahi memorial hospital showed that 12.5% of *P. aeruginosa* and 14.3% of *E. coli* were also multidrug resistant. There was no multidrug resistant isolates among *E. coli* from St. Luke's Anglican hospital but 30.8% of *P. aeruginosa* were multidrug resistant isolates.

Conclusion: The result of this study indicates that inanimate surfaces near infected patients and those frequently touched surfaces within the hospital environment were contaminated by *Pseudomonas aeruginosa* and *Escherichia coli*. Therefore, there is need for better improved sanitation in the study sites with concerted efforts in screening and monitoring occurrence of *Pseudomonas aeruginosa* from time to time.

Keywords: Surfaces; pathogens; antibiotics; susceptibility; resistant.

1. INTRODUCTION

In the past most nosocomial infections were caused by gram positive microbes in which *Staphylococcus aureus* was the primary cause of nosocomial infection [1]. Gram negative bacteria, such as *E. coli* and *Pseudomonas aeruginosa* that has the ability to cause opportunistic skin infections are also the major cause today [2]. The degree of occurrence of one or two of these organisms over others depends on the environment [3,4]. Environment significantly influences multiple factors in the chain of infection [5]. The transmission of microorganisms from environmental surfaces to patients is largely via contact with the surfaces [6]. Contamination of hospital equipment, medicines, and water supplies with hospital pathogens is a well-recognized cause of common-source outbreaks of infection. This can be especially troublesome in hospital environments where patients with immunodeficiency are at enhanced risk for contracting nosocomial infections [7,3]. *Pseudomonas aeruginosa* is capable of multiplying in two days, it can swim from one site to the next as motile cell or it multiplies as an adherent microcolony biofilm by producing a slimy layer [8] and [9].

Pal et al. [10] reported that *Pseudomonas aeruginosa* can last for longer period of time in moist surfaces such as suction apparatus, tap, operation theatre wards as well as sink. Nosocomial pathogens shed by patients can contaminate surfaces in hospitals at concentrations sufficient for transmission. It is constantly reintroduced into the hospital

environment by visitors and patients transferred from other facilities. Spread occurs from patient to patient on the hands of hospital personnel, by direct patient contact with contaminated reservoirs, and by the ingestion of contaminated foods and water. *Pseudomonas aeruginosa* has been recognized as an emerging opportunistic pathogen of clinical relevance i.e. it causes infections among immuno-compromised patients [4]. *Pseudomonas aeruginosa* has an abundance of virulence factors, including flagella, pili, lipopolysaccharides, alginate, alkaline protease, elastase, phospholipase C, exotoxin A, quorum sensing mechanisms, type III secretion system, pyocyanin, pyoverdine, and produces a number of toxic proteins which not only cause extensive tissue damage, but also interfere with the human immune system's defence mechanisms [4].

Escherichia coli is an important cause of urinary tract infections (UTIs). The systemic infections include bacteraemia, nosocomial pneumonia, cholecystitis, cholangitis, peritonitis, cellulitis, osteomyelitis, and infectious arthritis [11]. The presence of this microbe on surfaces in hospital could be as a result of faecal contamination. *Escherichia coli* O157:H7 infections occur worldwide; infections have been reported on every continent and are transmitted by the faecal-oral route. They can be spread between animals by direct contact and has the ability to convey virulence genes among microbes in a particular environment through horizontal gene transfer [12].

Drug resistance in *P. aeruginosa* may be mediated via several mechanisms. The

resistance mechanisms include; production of β -lactamases, efflux pumps and target-site or outer membrane modification [13]. More than 70% of these pathogens from the hospital environment are resistant to drugs or multi-drugs which are now the most leading cause of human death worldwide [14]. The occurrence of multi-drug resistance in hospital-associated pathogens has resulted in the emergence and re-emergence of difficult-to-treat nosocomial infections in patients [15]. Antibiotic-resistant *E. coli* may also pass on the genes responsible for antibiotic resistance to other species of bacteria, such as *Staphylococcus aureus*, through a process called horizontal gene transfer. Through the horizontal gene transfer *Escherichia coli* often carry multiple drug-resistance plasmids, and under stress, readily transfer those plasmids to other species [13].

Therefore, it is imperative to determine the distributions of the pathogens on surfaces in hospitals [16], because hospital is not only a place where sick people recover from their sickness but also where the illnesses get complicated and healthy people get infected. Whenever clinical procedures are performed, clients are at risk of infection during and after the procedures [6]. The aim of this study is to determine the prevalence and antibacterial susceptibility of some nosocomial pathogens isolated from some hospital environment in Zaria, Kaduna state.

2. MATERIALS AND METHODS

2.1 Study Area

The study area of this work encompass to hospitals in Zaria including St Luke Anglican hospital, Wusasa located at 11° 04' N and 007° 40' E and Major Ibrahim B. Abdullahi memorial hospital which is the second hospital selected for this study is situated at 11°06' N and 007° 41' E all at Greenwich meridian. These areas were located using Taiwan made Etrex® high-sensitive geographic positioning system (GPS) receiver.

2.2 Ethical Approval

The ethical approval was obtained from ethical committee of Kaduna state Ministry of Health and was used for sampling. Approval was also obtained from Medical Director, St. Luke's Hospital, Wusasa.

2.3 Inclusion Criteria

All surfaces which are easily contaminated through direct contact by health personnel and the subjects who also come in contact with the surfaces were included in the study.

2.4 Exclusion Criteria

All surfaces not directly contaminated by the health personnel and subjects who do not come in contact with the hospital surfaces were excluded.

2.5 Collection of Sample

The total number of samples collected for this study was 310 and all samples were collected in the morning before commencement of work in each hospital and hand swab of the staff were collected during working hours. Samples for the studies were collected from hands of some of the hospital staff, floors, toilets seats, operation tables, door knobs/door handles, nurses' table tops, bedrails, stretchers, cupboards, sinks, using sterile swab sticks using sterile cotton swabs wetted with sterile peptone water and transported to laboratory in screw capped plastic container and placed inside a transport box.

2.6 Bacterial Isolation and Purification

Each sample swab was inoculated into prepared sterile bacteriological peptone water and incubated at 37°C for 24 h for enrichment after which the turbid broth was sub cultured into solid differential media such as Eosin methylene blue agar (EMB), MacConkey agar plates for the isolation of *Escherichia coli* and *Pseudomonas centrimide* selective agar for *Pseudomonas aeruginosa*, all the plates were incubated at 37°C for 24 h. Discrete colonies were further sub cultured onto fresh prepared plates of the differential and selective media. The purified cultures were gram stained and stored on nutrient agar slants for biochemical and sugar fermentation tests and identification.

2.7 Biochemical Tests

The biochemical tests carried out for the identification were citrate and oxidase tests for identification of *Pseudomonas aeruginosa*, and indole, methyl red (MR), Voges Prokauer (VP) and citrate tests for identification of *Escherichia coli*. Further biochemical (or sugar fermentation) tests were carried out using Microgen Gram

negative-identification (GN-ID) system which comprises of two separate microwell test strips; GN A and GN B each strips contains 12 standardized biochemical dehydrated substrates in which isolates were introduced. Gram negative GNA for *E. coli* and GNA + GNB for *P. aeruginosa*. The identification of each pathogen was carried out according to the manufacturer's instructions.

2.8 Serology Test for Screening for *E. coli* O157:H7

The isolates of *Escherichia coli* were cultured for 24 h at 37°C on Sorbitol MacConkey agar plate and were used to carry out serology test for the identification of *E. coli* O157:H7 according to manufacturer's instruction.

2.9 Antibiotic Susceptibility Test

The antimicrobial susceptibility pattern was determined using Kirby-Bauer-CLSI modified single disc diffusion technique [17]. The antibiotic discs used were ampicillin (10 µg), vancomycin (30 µg), tetracycline (30 µg), cefoxitin (30 µg), chloramphenicol (30 µg), imipenem (10 µg), ceftazidime (30 µg), linezolid (10 µg) and gentamicin (10 µg), all the discs were obtained from Oxoid England and all the results of the antimicrobial susceptibility were interpreted using [18]. The dilution of each of the suspension of the 24 h test organisms and the standard isolates

(*Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 9027) were prepared using 0.5 scale of MacFarland's standard (1.5×10^8 cells/ml) [19]. The cell suspensions was inoculated by streaking on prepared Mueller-Hinton agar using sterile swab stick, then the antibiotic disc was placed on the inoculated medium aseptically with help of sterile forceps and incubate at 37°C for 24 h. The zones of inhibition by each of the antibiotics against the test organisms and the standard strains as positive control were measured and the result was interpreted.

3. RESULTS

The total number of isolates from Major Ibrahim B. Abdullahi memorial hospital presented in Fig. 1 was 23; of which 16 (69.6%) and 7 (30.4%) were *P. aeruginosa* and *E. coli* respectively. The total number of isolates from St Luke Anglican hospital was 21; of which *P. aeruginosa* and *E. coli* were 13 (61.9%) and 8 (38.1%) respectively (Fig. 1). The prevalence of the two pathogens isolated from surfaces at Major Ibrahim B. Abdullahi memorial hospital as presented in Table 1 shows that 33.3, 100, 50.0 and 85.7% of *P. aeruginosa* was isolated from toilet seats, sinks, stretchers and floors respectively and *Escherichia coli* was isolated from some nurses' table/staff table (40.0%), door knob (25.0%), and toilet seat (66.7%).

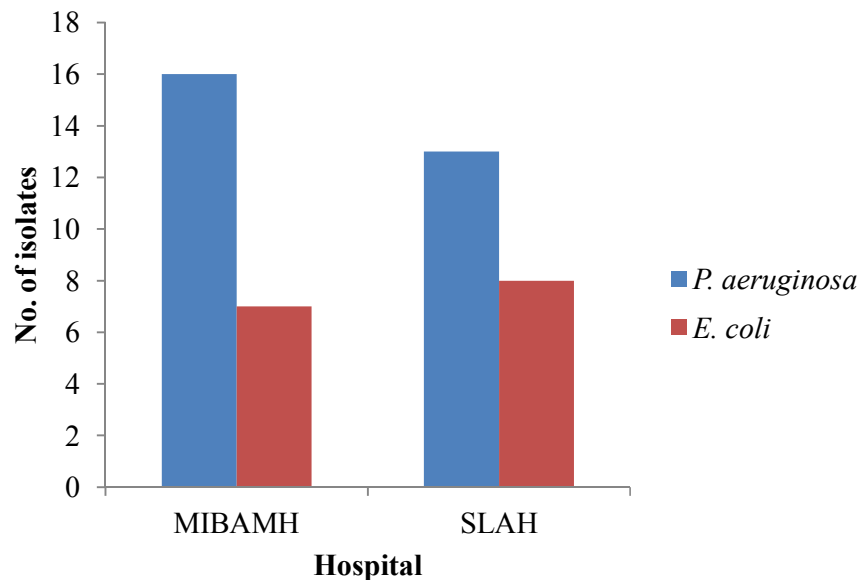


Fig. 1. Occurrence of bacterial isolates in the two hospital environment

Table 2 shows the distribution of the three pathogens on surfaces at St. Luke's Anglican hospital. *Pseudomonas aeruginosa* was isolated in this hospital from operation table (100%), sink (100%), stretcher (33.3%), floor (83.3%) and bedrail (30.0%). The total percentage distribution of *P. aeruginosa* was 27.0%. *Escherichia coli* was isolated from some door knob (42.9%), toilet seat (100%), floor (16.7%), bedrail (10.0%) and total prevalence of this pathogen isolated from this hospital was 16.7%. The result of serotyping of *E. coli* as shown in Table3 showed that all the eight (8) isolates of the *Escherichia coli* from St. Luke's Anglican hospital were negative (0%) for *E. coli* O157:H7 and all the isolate of *E. coli* from Major Ibrahim B. Abdullahi memorial hospital, none were *E. coli* O157:H7.

The antibiotic susceptibility profile of isolates from Major Ibrahim B. Abdullahi memorial hospital as in Table4 showed that some of the isolates of *P. aeruginosa* were resistant to tetracycline (43.8%) and chloramphenicol (12.5%), some were also susceptible to gentamicin (75.0%), imipenem (56.2%), chloramphenicol (50.0%), ceftazidime (68.7%) and tetracycline (56.2%). Of the isolates of *E. coli* 71.4 and 14.3% were resistant to tetracycline and ampicillin respectively. All the isolates of *E. coli* were 100% susceptible to gentamicin, cefoxitin ceftazidime and chloramphenicol; others were susceptible to ampicillin (57.1%) and tetracycline (28.6%).

The antibiotic susceptibility profile of pathogens from St. Luke's Anglican hospital as presented in Table 5 showed that of the total number of isolates of *P. aeruginosa* from this hospital a high percentage of 61.5% were resistant to tetracycline and 30.8% were resistant to chloramphenicol. All the isolates of *P. aeruginosa* were 100% susceptible to ceftazidime and imipenem. Some of them were susceptible to tetracycline (38.5%), chloramphenicol (30.8%) and gentamicin (61.5%). All the isolates of *E. coli* were 100% susceptible to ceftazidime, Chloramphenicol and gentamicin. Some were susceptible to ampicillin (62.5%), tetracycline (50.0%) and cefoxitin (75.0%).

Fig. 2 shows the percentage distribution of the total multidrug resistant strains of the two pathogens from the three hospitals. The multidrug resistant of the isolates from Major Ibrahim B. Abdullahi memorial hospital showed that 12.5% of *P. aeruginosa* and 14.3% of *E. coli* were also multidrug resistant. There was no

multidrug resistant isolates among *E. coli* from St. Luke's Anglican hospital but 30.8% of *P. aeruginosa* were multidrug resistant isolates.

4. DISCUSSION

This finding corroborates earlier report of Hassan et al. [20] and Page et al. [21] that surfaces in the hospital can act as reservoirs of microbes which could in turn lead to the spread of infection upon being touched, by healthcare workers, patients or visitors. Crowded conditions within the hospital, frequent transfer of patients from one unit to another, and concentration of patients highly susceptible to infection in one area (e.g. newborn infants, burn patients, and intensive care) all may contribute to development of nosocomial infections due to contaminated surfaces. Microbial flora may contaminate surfaces of objects, devices, and materials which subsequently contact susceptible body sites of patients [3]. The role of hospital environment in the distribution of nosocomial pathogen cannot be overemphasized.

The occurrence of *E. coli* on door knobs/door handles in Major Ibrahim B. Abdullahi memorial hospital and St. Luke's Anglican hospital respectively. The isolation of *E. coli* from the door knobs/door handles confirms the early report of Nworie et al. [22] for Abuja metropolis that that *E. coli* is one of the major nosocomial pathogens isolated from toilet seats in hospital environment. The contamination of door knob/door handle can be as a result of poor hand hygiene after using toilet. Bhalla et al. [23] and [24] reported that environmental contamination in healthcare settings occur when healthcare workers touch the surfaces with their hands or gloves especially after their routine patients care or when the patients come in direct contact with the surfaces. This finding also confirmed the reports of [25] and [26] that the hands of healthcare workers play an important role in the propagation of microorganism within the healthcare environment and ultimately to the patients if not properly wash and disinfected. WHO [25] reported that despite the hand washing and disinfection, many studies continue to show poor compliance with hand hygiene in most hospitals. If hand hygiene practices are suboptimal, microbial colonisation is more easily established and/or direct transmission to patients or a fomites in direct contact with the patients or fomites [27]. It has been reported that organisms are capable of surviving on hands of health care workers for at least several minutes following contamination

[27]. *Pseudomonas aeruginosa* was not isolated from door knobs/door handles of the two hospitals. Therefore, hand hygiene is a constant concern and the promoting of it is necessary as part of each hospital's infection-control and safety programs.

High occurrence of *P. aeruginosa* on toilet seat, sink, floors, operation table and stretchers and *E. coli* from toilet seats, door knobs/door handles confirmed the report of [7,3,28,16,29]. *Pseudomonas aeruginosa* and *E. coli* are among the major contaminants of hospital surfaces such

Table 1. Prevalence (%) of the isolates in Major Ibrahim B. Abdullahi Memorial Hospital

Sample source	Sample size	Total positive isolates	Total % of isolates	<i>P. aeruginosa</i> (%)	<i>E. coli</i> (%)
NHS	11	-	-	-	-
NTT/ST	9	2	55.6	-	2
DK/DH	25	2	32.0	-	2
TS	8	3	37.5	1	33.3
OT	4	2	50.0	2	100
Sink	14	5	35.7	5	100
Stretcher	14	2	28.6	2	50.0
Floor	35	6	20.0	6	85.7
BR	19	-	-	-	-
CB	16	1	18.8	-	1
Total	155	23	29.7	16	34.8

KEY: SHS = Nurses' hand swab, NTT/ST = Nurses table top/staff table, DK/DH = Door knob/Door handle, TS= Toilet seat, OT= Operation table, BR = Bedrail, CB = Cup board

Table 2. Prevalence (%) of the isolates in St. Luke Anglican Hospital

Sample source	Sample size	Total positive isolates	Total % of isolates	<i>P. aeruginosa</i> (%)	<i>E. coli</i> (%)
NHS	15	-	-	-	-
NTT/ST	16	-	-	-	-
DK/DH	20	3	35.0	-	3
TS	7	3	42.9	-	3
OT	6	1	16.7	1	100
Sink	10	3	30.0	3	100
Stretcher	10	1	30.0	1	33.3
Floor	30	6	20.0	5	83.3
BR	22	4	45.5	3	30.0
CB	19	-	-	-	-
Total	155	21	31.0	13	27.0

KEY: SHS = Nurses' hand swab, NTT/ST = Nurses table top/staff table, DK/DH = Door knob/Door handle, TS= Toilet seat, OT= Operation table, BR = Bedrail, CB = Cup board

Table 3. The serotype of *Escherichia coli* for *E. coli* O157:H7

Hospital	Number of isolates	Test latex	Control text	Interpretation	Total number of <i>E. coli</i> O157:H7 identified
SLAH	8	-	-	<i>E. coli</i> O157:H7 not present	0
MIBAMH	7	-	-	<i>E. coli</i> O157 not present	0

KEY: MIBAMH = Major Ibrahim B. Abdullahi Memorial Hospital, SLAH = St Luke's Anglican Hospital

Table 4. Antibiotic profile of the three isolates from Major Ibrahim B. Abdullahi Memorial Hospital

Antibiotic	<i>Pseudomonas aeruginosa</i> (N = 16)			<i>Escherichia coli</i> (N = 7)		
	R	I	S	R	I	S
AMP (10 µg)		NT		1(14.3%)	2(28.6%)	4(57.1%)
TE (30 µg)	7(43.8%)	0(0.0%)	9(56.2%)	5(71.4%)	0(0.0%)	2(28.6%)
CAZ (30 µg)	0(0.0%)	5(31.3%)	11(68.7%)	0(0.0%)	0(0.0%)	7(100%)
C (30 µg)	2(12.5%)	6(37.5%)	8(50.0%)	0(0.0%)	0(0.0%)	7(100%)
IMP (10 µg)	0(0.0%)	7(43.8%)	9(56.2%)		NT	
FOX (30 µg)		NT		0(0.0%)	0(0.0%)	7(100%)
CN (30 µg)	0(0.0%)	4(25.0%)	12(75.0%)	0(0.0%)	0(0.0%)	7(100%)

Where: FOX = Cefoxitin, AMP =Ampicillin, CN = Gentamicin, TE = Tetracycline, IMP = Imipenem, CAZ = Ceftazidime, R = Resistant, C = Chloramphenicol, I = Intermediate, NT = Not Tested, S= sensitive

Table 5. Antibiotic profile of the three isolates from St. Lukes' Anglican Hospital

Antibiotic	<i>Pseudomonas aeruginosa</i> (N = 13)			<i>Escherichia coli</i> (N = 8)		
	R	I	S	R	I	S
AMP (10µg)	NT			1(12.5%)	2(25.0%)	5(62.5%)
TE (30µg)	8(61.5%)	0(0.0%)	5(38.5%)	2(25.0%)	2(25.0%)	4(50.0%)
CAZ (30µg)	0(0.0%)	0(0.0%)	13(100%)	0(0.0%)	0(0.0%)	8(100%)
C (30µg)	4(30.8%)	5(38.4%)	4(30.8%)	0(0.0%)	0(0.0%)	8(100%)
IMP (10µg)	0(0.0%)	0(0.0%)	13(100%)	NT		
FOX (30µg)	NT			0(0.0%)	2(25.0%)	6(75.0%)
CN (30µg)	0(0.0%)	5(38.5%)	8(61.5%)	0(0.0%)	0(0.0%)	8(100%)

Where: FOX = Cefoxitin, AMP =Ampicillin, CN = Gentamicin, TE =Tetracycline, CAZ = Ceftazidime, IMP = Imipenem, R = Resistant, C = Chloramphenicol, I = Intermediate, S = Sensitive, NT = Not Tested

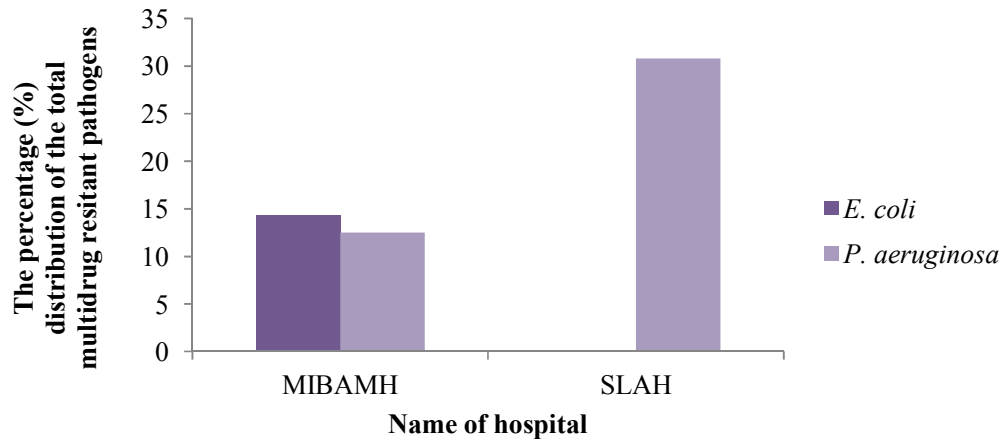


Fig. 2. The percentage multidrug-resistant isolates from the two hospital environment

KEY: MIBAMH = Major Ibrahim B. Abdullahi Memorial Hospital, SLAH = St Luke's Anglican Hospital

as room, door handles, sinks, toilet seats, floors, operation tables, sterile packaging, mops, ward fabrics and plastics, hands of healthcare workers, keyboards and taps, stethoscopes and telephones. The high level of contamination of these pathogens could also be as a result of inadequate decontamination of the microbial load

from the surfaces [30]. The 100% prevalence of *P. aeruginosa* in sinks of the two hospitals is also higher compare with a work reported by [10] that 4.47% of the pathogen was isolated from sinks in hospital environment in Iran. *P. aeruginosa* is predominantly found on toilet seats, sinks and other moist environment in hospitals and may be

seeded (forming biofilm) into toilets and remain on the toilet seats for a long time even after multiple flushing and cleaning with antimicrobial agents as reported by [31]. Also, the prevalence rate of *P. aeruginosa* on operation table in St Lukes' hospitals is higher compared to a work reported by [10] that 9.6% of the pathogen was isolated from operation table in a hospital in India. This may be as a result of improper disinfection of the operation table or inadequate removal of moist after operation.

The resistance of some *P. aeruginosa* to the some antibiotics in this research shows that *P. aeruginosa* as reported by [9] is resistant to most antibiotics because it is found naturally in soil; it has developed many resistances to naturally occurring antibiotics produced by bacilli, actinomycetes and moulds and their resistance to most antibiotics is attributed to efflux pumps which pump out some antibiotics before the antibiotics are able to act. The 0.0% of the antibacterial resistance profile of *E. coli* to gentamicin, cefoxitin, ceftazidime and chloramphenicol in this finding is in agreement with research findings of [32] in Sudan, who also found that *E. coli* was 0.0% resistant to gentamicin, cefoxitin, ceftazidime and chloramphenicol. The resistance of *E. coli* to ampicillin could be as a result of production of β -lactamase enzyme which has the ability to deactivate the efficacy of this β -lactam drug as reported by [33]. In this research, gentamicin, cefoxitin, ceftazidime and chloramphenicol were the most active antibiotics against *E. coli*.

The multidrug resistance of *P. aeruginosa* from Major Ibrahim B. Abdullahi memorial hospital (12.5%) and St. Luke's Anglican hospital (30.8%) confirms the report of [34] that outbreaks of multidrug-resistant *P. aeruginosa* colonization or infection occurred in urology wards, a burn unit, haematology/oncology units, and adult and neonatal critical care units and that various medical devices and environmental reservoirs was implicated in the outbreak including antiseptic solutions and lotions; endoscopy equipment; ventilator apparatus, sink and hand swab. *Pseudomonas aeruginosa* has been increasingly recognized for its ability to cause significant hospital-associated outbreaks of infection, particularly since the emergence of multidrug resistant strains. The occurrence of multi-drug resistance is very common especially among the Gram negative bacteria. Multidrug resistant *E. coli* are widely distributed in hospitals and are increasingly being isolated from

community. Thus, there is urgent need to find out new antimicrobial agents [35].

5. CONCLUSION

The result of this study indicated that inanimate surfaces near infected patients and those frequently touched surfaces within the hospital environment were contaminated by *Pseudomonas aeruginosa* and *Escherichia coli*. These surfaces can serve as reservoirs of these pathogens. Some of the pathogens were resistant and multi-resistant to the antibiotics used in this study. Therefore, there is need for better improved sanitation in the study sites with concerted efforts in screening and monitoring occurrence of *Pseudomonas aeruginosa* from time to time.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Hammuel C, Idoko MO, Migap HH, Ambrose N. Occurrence and antibiogram profile of *Staphylococcus aureus* isolated from some hospital environment in Zaria, Nigeria. *Afric J. Microbio. Res.* 2015;9(19):1304-1311.
2. Qayyum S, Sattar A, Waqas B. Hospital Acquired infections: Knowledge about and its prevention. *J. Prof. Med.* 2010;17(2):168-173.
3. Chikere CB, Omoni VT, Chikere BO. Distribution of potential nosocomial pathogens in a hospital environment. *Afric. J. of Biotech.* 2008;7(20):3535-3539.
4. Okonko IO, Soley FA, Amuson TA, Ogun AA, Ogunnusi TA, Ejembi J. Incidence of multi-drug resistance (MDR) organisms in Abeokuta, South-western Nigeria. *Glob. J. Pharmacol.* 2009;3(2):69-80.
5. Ekrami A, Kayedani A, Jahangir M, Kalantar E, Jalali M. Isolation of common aerobic bacterial pathogens from the environment of seven hospitals. *Ahvaz, Iran Jundishapur J. Microbio.* 2011;4(2):75-82.
6. Kampf G, Kramer A. Epidemiologic background of hand hygiene and evaluation of the most important agents for scrubs and rubs. *J. Clin. Microbio. Revol.* 2004;17:863-893.

7. Jarvis WR. The inanimate environment. In: Bennet JV, Bracman PS (eds), Hospital infections, 5th ed. Philadelphia, PA, Lippincott-Raven. 2007;275-302.
8. Hardy JK. A study of the relationship between environmental contamination due to Methicillin-resistant *Staphylococcus aureus* (MSRA) and patients' acquisition of MRSA. J. Infec. Control. Hosp. Epidemiol. 2006;27:127-132.
9. Harris DD, Pacheco A, Lindner AS. Detecting potential pathogens on hospital surfaces: An Assessment of carpet tile flooring in the hospital patient environment. Journal of Indoor and Built Environment. 2010;19(2):239-249.
10. Pal RB, Rodrigues M, Datta S. Role of *Pseudomonas* in nosocomial infections and biological characterization of local strains. J. Biosci. Tech. 2010;1(4):170-179.
11. Buchanan RL, Doyle, MP. Food borne disease significance of *Escherichia coli* O157:H7 and other Enterohemorrhagic *Escherichia coli*. J. Food Tech. 1997; 51(10):69-76.
12. Alam MJ, Zurek L. Seasonal prevalence of *Escherichia coli* O157:H7 in beef cattle faeces. J. Food Protect. 2006;69(12):3018-3020
13. Tavajjohi Z, Moniri R. Detection of ESBLs and MDR in *Pseudomonas aeruginosa* in a tertiary-care Teaching Hospital. Iran. J. Clin. Infect. Dis. 2011;6(1):18-23
14. Anton YP, David CP. Hospital Acquired infection due to Gram-negative bacteria. The new Eng. J. Med. 2010;362:19.
15. Hammuel C, Jatau ED, Whong CMZ. Prevalence and antibiogram pattern of some nosocomial pathogens isolated from hospital environment in Zaria, Nigeria. Aceh Int. J. Sc. Tech. 2014;3(3):131-139.
16. Dwivedi M, Mishra A, Singth RK, Azim A, Baronia AK, Prasad KN. Nosocomial cross-transmission of *Pseudomonas aeruginosa* between patients in a tertiary intensive care unit. Ind. J. Path. Microbio. 2009;52:509-513.
17. Cheesborough M. District Laboratory manual in Tropical countries. Low price edition. Cambridge University Press. 2005;156-158.
18. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. Twenty-Third Information Supplement. 2013;33(1):44-128.
19. Coyle MB. Manual of antimicrobial susceptibility testing. American Society of Microbiology press, Washinton D.C. 2005;25:39.
20. Hassan AN, Birt DM, Frank JF. Behaviour of *Listeria monocytogenes* on *Pseudomonas putida* biofilm on a condensate forming surface. J. Food Protect. 2004;67(2):322-327.
21. Page K, Wilson M, Parkin IP. Antimicrobial surfaces and their potential in reducing the role of the inanimate environment in the incidence of hospital-acquired infections. J. Materi. Chemi. 2009;1-23.
22. Nworie A, Ayeni JA, Eze UA, Azi SO. Bacterial contamination of door handles/knobs in selected public conveniences in Abuja metropolis, Nigeria: A public health threat. Conti. J. Med. Res. 2012;6(1):7-11.
23. Bhalla V, Pultz NJ, Gries DM, et al. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalised patients. J. Infec. Control Hosp. Epidemio. 2004;25: 164-167.
24. Boyce JM. Environmental contamination makes an important contribution to hospital infection. J. Hosp. Infect. 2007;65(2):50-54.
25. WHO. Guidelines for hand hygiene in health care (Advanced draft). Geneva: World Health Organization; 2006.
26. Hayden MK, Donald MD, Blom W, et al. Risk of hand or glove contamination after contact with patients colonized with vancomycin-resistant *Enterococcus* or the colonized patients' Environment. J. Infec. Control Hosp. Epidemio. 2008;29(2):149-154.
27. Allegranzi B, Pittet D. Role of hand hygiene in healthcare-associated infection prevention. J. Hosp. Infect. 2009;73:305-315.
28. Wren MWD, Rollins MSM, Jeanes A, Hall TJ, Coen PG, Gant VA. Removing bacteria from hospital surfaces: A laboratory comparison of ultra-microfiber and standard clothes. J. Hosp. Infect. 2008;70:265-271.
29. Ijioma BC, Kalu IG, Nwachukwu CU. Bacteria prevalence on the Environmental labour as the cause of nosocomial infections in General hospital Umuguma and Umezuruike hospital Owerri, in Imo State, Nigeria. Report and Opinion. 2010;2(9):45-52.

30. Addy PAK, Antepim G, Frimpong EH. Prevalence of Pathogenic *Escherichia coli* and parasites in infants with diarrhoea in Kumasi, Ghan. East Africi. Med. J. 2004;81(7):353-357.
31. Udeze AO, Adeyemi AT, Adeniji FO, et al. Plasmid mediated ampicillin resistant bacteria isolates from University of Ilorin Health Centre. New York Sci. J. 2012;5(4):56-63.
32. Mukhtar AM, Saeed HA. Profile of antibiotic sensitivity and resistance of some pathogenic bacteria isolated from clinical specimens in Sudan. J. Sci. Tech. 2011;12(1):14-19.
33. Hassan SA, Jamal SA, Kamal M. Occurrence of multidrug resistant and ESBL producing *E. coli* causing urinary tract infections. J. Basic Applied Sci. 2011;7(1):39-43.
34. Hota S, Hirji Z, Stockton K, et al. Outbreak of multidrug-resistant *Pseudomonas aeruginosa* colonization and infection secondary to imperfect intensive care unit room design. J. Infec. Control Hosp. Epidemio. 2009;30(1):25-33.
35. Ibrahim ME, Bial NE, Hamid ME. Increased multidrug resistant *E. coli* from hospitals in Khartoum, Sudan. J. Health Sci. 2012;12(3):368-375.

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