World Applied Sciences Journal 20 (6): 766-775, 2012
ISSN 1818-4952

DOI: 10.5829/idosi.wasj.2012.20.06.667

Plasmid Profile Analysis of Multidrug Resistant *Pseudomonas aeruginosa* Isolated from Wound Infections in South West, Nigeria

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Abstract: *Pseudomonas aeruginosa* has become increasingly recognized as an emerging opportunistic pathogen of clinical relevance. A total of 110 *Pseudomonas aeruginosa* was obtained from clinical wound samples in three tertiary hospitals in South West, Nigeria. Isolated pure cultures of bacteria were subjected to various morphology and biochemical tests. After which they were identified using Bergey's Manual of Systematic Bacteriology. One hundred and ten (110) *Pseudomonas aeruginosa* isolated from clinical wounds were subjected to antibiotic susceptibility testing by disk diffusion and plasmid profiling. Fifteen (15) different antibiotics discs were used to determine the drug sensitivity pattern of the isolates. Plasmids were extracted by the alkaline lysis method (Zymogen UK). Electrophoresis of the DNA was carried out on a 0.8% agarose gel. Variation occurred in multiple antibiotic resistance patterns among various strains of *Pseudomonas aeruginosa* isolated from the clinical wound samples. The antibiotic resistant pattern showed that *Pseudomonas aeruginosa* had high resistant to amoxicillin 92.7%, ampicillin 90%, cloxacillin 88.2%, cotrimoxazole 77.3%, erythromycin 72.7%, tetracycline 70.9%, streptomycin 65.5% and ofloxacin 60% and had low resistant to ceftazidime (20%), gentamycin (26.4%), levoxin (30.9%), ceftriaxone (34.5%) and ciprofloxacin (35.5%). Plasmid profile was carried out on 22 selected multi drug resistant (MDR) isolates that were resistant to three or more classes of antibiotics. Eight (36.4%) strains were found to possess plasmid bands. Six of the strains had single plasmid band while two strains possessed two bands with sizes ranging from 662 to 830bp. The sizes of the plasmids among *P. aeruginosa* isolates ranged from 662bp to 830bp. All the strains that had plasmids were resistant to amoxicillin, ampicillin, cloxacillin, cotrimoxazole, erythromycin and tetracycline.

Key words: Antibiotics · Electrophoresis · *P. aeruginosa* · Multi Drug Resistance (MDR) · Plasmid Profile · Nigeria

INTRODUCTION

Pseudomonas aeruginosa is one of the most common causes of burn wound infections [1]. *P. aeruginosa* is the most common pathogen causing wound infection compatible with other reports, especially from developing countries [2, 3]. *Pseudomonas aeruginosa* is an epitome of opportunistic nosocomial pathogen, which causes a wide spectrum of infections and leads to substantial morbidity in immune compromised patients. Despite therapy the mortality due to nosocomial *Pseudomonas pneumonia* is approximately 70%. Unfortunately, *Pseudomonas aeruginosa* develops resistance to most of antibiotics thereby jeopardizing the selection of appropriate treatment [4].

In developing countries, large number of people die daily of preventable and curable diseases such as wound infections. Wound infection is one of the health problems

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that are caused and aggravated by the invasion of pathogenic organisms in different parts of the body. Previous studies from different parts of the country showed that *Pseudomonas species, Staphylococcus aureus, Klebsiella species, Escherichia coli, Proteus species, Streptococcus species, Enterobacter species* and coagulase negative staphylococci are the most common pathogens isolated from wound [5]. The wound sometimes gets infection by either single or multiple organisms. Wound infections are mostly due to nosocomial pathogens that differ from country to country and from hospital to another within the same region, which remains the major source of post-operative morbidity [5].

Most pseudomonads known to cause disease in humans are associated with opportunistic infections. These include *P. fluorescence, P. putida, P. cepacia, P. stutzeri, P. maltophila and P. putrefaciens.* Only two species, *P. mallei* and *P. pseudomallei*, produce specific human disease: glanders and meliodosis. *Pseudomonas aeruginosa* ever growing multi drug resistance has been widely reported [6]. This cuts across the third and fourth generation cephalosporins, the generic fluoroquinolones, the aminoglycosides and the advanced beta-lactam antibiotics [7].

Pseudomonas aeruginosa is naturally resistant to β-lactams, including broad-spectrum cephalosporins, quinolones, chloramphenicol and tetracyclines, mainly because of the very low permeability of their cell wall. Moreover, *P. aeruginosa* is characterized by the production of inducible cephalosporinase, active efflux and poor affinity for the target (DNA gyrase), three mechanisms that synergize with poor cell wall permeability [8]. Once colonization and infection are established, *P. aeruginosa* becomes one of the worst pathogens of human and it is known to possess intrinsic multi-drug resistance capabilities [6]. In its large genome of 6.3 million base pairs (bp) houses 8 virulence genes are identified. Moreover, the large genome size increases the probability of possible mutation sites and thus gives reasons for its virulence versatility, its growing multi drug resistance and the high mortality rate associated with its infection [6].

A high rate of spread of resistant gene has been suspected as the cause of increased antibiotic resistance cases in it. Plasmid carry genes that could be spread by conjugation and transduction while the genome based resistant genes are also spread by replication. Intrinsic and acquired antibiotic resistance makes *P. aeruginosa* one of the most difficult organisms to treat. The high intrinsic antibiotic resistance of *P. aeruginosa* is due to several mechanisms: a low outer membrane permeability, the production of an AmpC β-lactamase and the presence of numerous genes coding for different multidrug resistance efflux pumps [9].

A high number of acquired resistance genes coding for aminoglycoside-modifying enzymes (AME) and β-lactamases have been noted in *P. aeruginosa* [10], extended-spectrum β-lactamases have been increasingly reported [11] and metallo-β-lactamases have also started to emerge in *P. aeruginosa* [12]. *Pseudomonas aeruginosa* is a classic opportunistic pathogen especially because of its innate resistance to many antibiotics and disinfectants; and also due to its armoury of putative virulence factors plus additional acquired resistance due to plasmids [13].

In Nigeria, a study from the South West showed that *Pseudomonas aeruginosa* had been isolated from urine (4.6%), reproductive tract (2.1%) and wound infections (16.3%) [14, 15]. Another study from the north reported the occurrence of *Pseudomonas aeruginosa* in urine samples to be 4.6% [16]. Another report from the south west isolated *Pseudomonas aeruginosa* from 39.3% in wound swabs, 41.9% in ear swabs. From the south, *Pseudomonas aeruginosa* was isolated from 41% of cases with discharging ears [15, 17].

Despite improvements in antibiotic therapy *Pseudomonas aeruginosa* is intrinsically resistant to a number of antimicrobial agents frequently including multiple classes of antimicrobial agents [15]. Subsequently outbreaks due to multi resistant *Pseudomonas aeruginosa* have been reported in various nosocomial settings, such as intensive care units (ICUs) [15]. Typing techniques useful for establishing clonal relationships between individual isolates in hospital settings are therefore important to recognize nosocomial transmission and guide infection control practice [15]. Typing techniques such as PFGE, SDS-PAGE and RAPDPCR have been found to be useful for epidemiological study of *Pseudomonas aeruginosa* [15, 18]. Plasmid profile analysis examines the total bacterial plasmid content, or subjects plasmids to restriction endonucleases and separates the cleaved plasmid DNA by electrophoresis for analysis. This method is a powerful tool for following the spread of antibiotic resistance, because resistance is usually passed between bacteria on plasmids [19].

This study was carried out to determine the antibiotic resistant patterns of *Pseudomonas aeruginosa* isolates obtained from clinical wound samples in south west, Nigeria against some of the commonly prescribed antibiotics and to determine the plasmid profile of the multiple antibiotic resistant strains.

MATERIALS AND METHODS

Bacteriology: Three hundred (300) clinical wound specimens were collected from three tertiary hospitals in South west Nigeria; cultured on blood agar and MacConkey agar plates and incubated at 37°C for 24 h and on Mueller Hinton agar plates to assess pigment production. The culture plates were processed using standard microbiological procedures [20]. Characterisation and identification of *P. aeruginosa* was carried out using a combination of colonial morphology, Gram stain characteristics and biochemical reactions [20]. A total of 110 *Pseudomonas aeruginosa* isolates were obtained from clinical wound samples collected from three tertiary hospitals in South west, Nigeria.

Antimicrobial Sensitivity Testing: Commercially available antimicrobial discs (Abtek Biological Ltd UK) were used to determine the drug sensitivity and resistance pattern of the isolates. The antibiotic sensitivity test of each isolate was carried out as described by the Kirby – Bauer disc diffusion method [21] as recommended by the National Committee for Clinical Laboratory Standards [22]. After incubation, the diameter of the zone of inhibition was measured and compared with zone diameter interpretative chart [22, 23] to determine the sensitivity of the isolates to antibiotics.

Plasmid Isolation and Profiling: Plasmid isolation was done using a commercial plasmid isolation kit (Plasmid Miniprep Kit, Zymogen Co. Ltd. UK) according to the manufacturer instructions.

Gel Electrophoresis: Electrophoresis of the DNA was carried out on a 0.8% agarose gel according to Bikandi *et al*. [24] procedure.

RESULTS

Table 1 shows the antibiotic resistant and susceptibility patterns of the 110 *Pseudomonas aeruginosa* isolates obtained from clinical wound samples. The 110 *Pseudomonas aeruginosa* isolates showed resistance to 15 different antibiotics; amoxicillin (92.7%), ampicillin (90.0%), cloxacillin (88.2%), cotrimoxazole (77.3%), erythromycin (72.7%), tetracycline (70.9%), streptomycin (65.5%), ofloxacin (60.0%), cefuroxime (54.5%) and augmentin (51.8%). Lower resistance to ceftazidime (20%), gentamycin (26.4%), levoxin (30.9%), ceftriaxone (34.5%) and ciprofloxacin (35.5%) was also recorded (Table 1).

Table 1: Antibiotic resistance patterns of the 110 *Pseudomonas aeruginosa* isolates

Class of Antibiotic	Type of Antibiotic	Number and Percentage of Resistant	Number and Percentage of Susceptible
Penicillin	Ampicillin (25mg)	99(90)	11(10)
	Amoxicillin (25mg)	102(92.7)	8(7.3)
	Augmentin (25mg)	57(51.8)	53(48.2)
	Cloxacillin (5mg)	97(88.2)	13(11.8)
Aminoglycoside	Streptomycin (10mg)	72(65.5)	38(34.5)
	Gentamycin (10mg)	29(26.4)	81(73.6)
Cephalosporin	Ceftazidime (30mg)	22(20.0)	88(80.0)
	Ceftriaxone (30mg)	38(34.5)	72(65.5)
	Cefuroxime (30mg)	60(54.5)	50(45.5)
Quinolones	Ciprofloxacin (10mg)	39(35.5)	71(64.5)
	Ofloxacin (30mg)	66(60.0)	44(40)
	Levoxin (25mg)	34(30.9)	76(69.1)
Macrolides	Erythromycin (10mg)	80(72.7)	30(27.3)
Tetracycline	Tetracycline (25mg)	78(70.9)	32(29.1)
Cotrimoxazole	Cotrimoxazole (25mg)	85(77.3)	25(22.7)

Table 2: Antibiotic profile of multi drug resistant *Pseudomonas aeruginosa* isolates detected from the clinical wound samples

Isolates	Resistant Pattern
P 4	Amp, Amx, Cxm, Cip, Cxc, Cot, Ery, Ofl, Str, Tet, Lev
P 9	Amp, Amx, Aug, Cef, Cxm, Cip, Cxc, Ery, Gen,
P 11	Amp, Amx, Cxm, Aug, Cef, Cxc, Cot, Ery, Gen, Lev,
P 15	Amp, Amx, Aug, Cef, Cot, Gen, Lev, Ofl, Str, Tet
P 26	Amp, Amx, Cef, Caz, Cxm, Cxc, Cot, Ery, Str, Tet, Lev
P 34	Amp, Amx, Aug, Cxm, Cxc, Cot, Ery, Ofl, Tet
P 39	Amp, Amx, Cxm, Cxc, Cot, Ery, Gen, Ofl, Str, Tet
P 45	Amp, Amx, Cxm, Cip, Cxc, Cot, Ery, Lev, Ofl, Str, Tet
P 46	Amp, Amx, Aug, Cxm, Cip, Cxc, Cot, Ery, Ofl, Tet
P 52	Amp, Amx, Aug, Cef, Cxm, Gen, Lev, Ofl, Str, Lev
P 54	Amp, Aug, Cef, Cxm, Cot, Ery, Gen, Lev, Ofl, Str,
P 60	Amp, Amx, Cef, Caz, Cxm,Cxc, Cot, Ery, Gen, Lev
P 63	Amp, Amx, Aug, Cxm, Cip, Cxc, Cot, Ery, Lev, Str, Tet
P 69	Amp, Amx,Cef, Caz, Cxm, Cxc, Cot, Ery, Ofl, Str, Tet
P 73	Amx, Aug, Cxm, Cxc, Cot, Ery, Ofl, Str, Tet, Lev
P 79	Amx, Aug, Cxm, Cip, Cxc, Ery, Ofl, Str, Tet, Lev
P 84	Amp, Amx, Cef, Cxm, Cxc, Cot, Ery, Gen, Ofl, Str, Tet
P 86	Amp, Amx, Cef, Cxm, Cxc, Cot, Ery, Ofl, Str, Tet
P 91	Amp, Amx, Aug, Cef, Cxm, Cot, Ery, Gen, Ofl,
P 99	Amp, Amx, Aug, Cef, Cip, Cxc, Cot, Ery, Gen, Ofl, Tet
P 105	Amp, Amx, Cef, Cxm, Cxc, Cot, Ery, Str, Tet
P 109	Amp, Amx, Caz, Cxm, Cip, Cxc, Ery, Ofl, Str, Lev

Gentamycin (Gen, 10mg), Erythromycin (Ery, 10mg), Ampicillin (Amp, 25mg), Augmentin (Aug, 25mg), Cotrimoxazole (Cot, 25mg), Tetracycline (Tet, 25mg), Streptomycin (Str, 10mg), Ciprofloxacin (Cip, 10mg), Cloxacillin (Cxc, 5mg), Amoxicillin (Amx, 25mg), Cefuroxime (Cxm, 30mg), Ceftriaxone (Cef, 30mg), Levoxin (Lev, 25mg) Ceftazidime (Caz, 30mg) and Ofloxacin (OFL, 30mg).

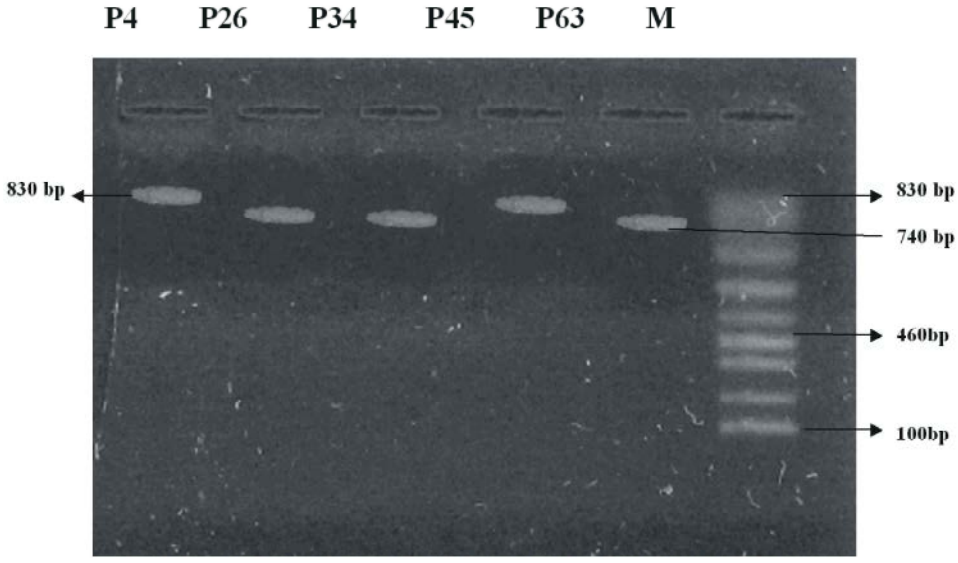

Plate 1: Plasmid profiles of the multi drug resistant *Pseudomonas aeruginosa* isolates: P4 (830bp), P26 (740bp), P34 (740bp), P45 (830bp) and P63 (740bp) is the clinical isolates. *Lane* M, 1000bp ladder.

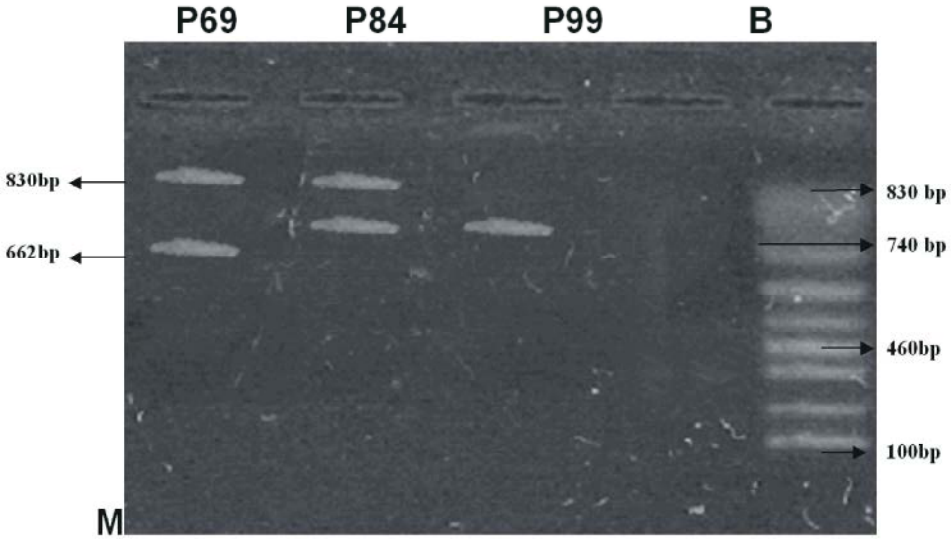

Plate 2: Plasmid profiles of the multi drug resistant *Pseudomonas aeruginosa isolates*: P69 (830bp, 662bp), P84 (830bp, 735bp), P99 (735bp). B (Negative control), *Lane* M, 1000bp ladder.

Antibiotic resistant profile revealed that twenty two *Pseudomonas aeruginosa* isolates were resistant to three or more classes of antibiotics in this study. The isolates were P4, P9, P11, P15, P26, P34, P39, P45, P46, P52, P54, P60, P63, P69, P73, P79, P84, P86, P91, P99, P105 and P109. Most of these strains were resistant to ampicillin, amoxicillin, cloxacillin, cotrimoxazole and erythromycin (Table 2).

The plasmid analyses revealed that there were detectable plasmids in 8(36.4%) out of the 22 selected multi-drug resistant *Pseudomonas aeruginosa* isolates. Fourteen of the isolates possessed no plasmids, six isolates possessed single sized plasmids (740bp - 830bp) while two strains of the isolates had two plasmids with sizes from (662bp - 830bp) (Plates 1 and 2). *Pseudomonas aeruginosa* isolates P4 and P45 had plasmid size of 830bp respectively. Three *Pseudomonas aeruginosa* isolates P26, P34 and P63 had plasmid size of 740bp respectively (Plate 1). In plate 2, two of the *Pseudomonas aeruginosa* isolates P69 and P84 had two plasmids with sizes of 830 & 662bp and 830 & 740bp respectively while *Pseudomonas aeruginosa* isolate P99 had plasmid size of 740bp.

DISCUSSION

This study examined the plasmid profile of multi drug resistant *Pseudomonas aeruginosa* in south west, Nigeria and found that the level of multidrug resistance to antibiotics in hospitals has increased, but was still relatively low compared to other reports from other countries [25-29]. *Pseudomonas aeruginosa* is currently one of the most frequent nosocomial pathogen and the infections due to this organism are often difficult to treat due to antibiotic resistance [25-29].

The overall incidence of antibiotic resistance of *Pseudomonas aeruginosa* isolates was high in this study. *Pseudomonas aeruginosa* had high resistant to amoxicillin 92.7%, ampicillin 90%, cloxacillin 88.2%, cotrimoxazole 77.3%, erythromycin 72.7%, tetracycline 70.9%, streptomycin 65.5% and ofloxacin 60%. In a study by Smith *et al*. [15], twenty *Pseudomonas aeruginosa* isolates showed resistance to 12 different antibiotics with six being 100% resistant and plasmids were detected in 16 (80%) of the isolates.

According to Li *et al*. [8], *Pseudomonas aeruginosa* is naturally resistant to β-lactams, including broad-spectrum cephalosporins, quinolones, chloramphenicol and tetracyclines, mainly because of the very low permeability of their cell wall but in this present study there was a good susceptibility for some of the groups of antibiotics. High susceptibility activities against *P. aeruginosa* were recorded in antibiotics such as ceftazidime 80.0, gentamycin 73.6 and levoxin 69.1%. This result is in agreement with a research carried out by Van Eldere [30] that reported ceftazidime and gentamycin as good antibiotics against *P. aeruginosa*.

As compared to other studies, in this study *Pseudomonas aeruginosa* showed reduced sensitivity to ciprofloxacin (64.5%). Ciprofloxacin has been stated to be the most potent drug available for the treatment of P. aeruginosa infections [31].

Generally most strains of *P. aeruginosa* are known to besensitive to ceftazidime [27]. *Pseudomonas aeruginosa* showed highest susceptibility to ceftazidime, a 3rd generation cephalosporin and this is consistent with reports from other groups of workers [27, 33]. However, *Pseudomonas aeruginosa* recorded very low susceptibility (45.5%) to cefuroxime another cephalosporin though of the 2nd generation. *P. aeruginosa* resistance activity observed in this research against cephalosporins ranged from 20 to 54.5%. This finding is relatively not in agreement to the one reported by Yetkin *et al*. [32] in 2006, in which the percentage of resistance to cephalosporins was in the range of 27 to 88%. In a study conducted in north central, Nigeria by Olayinka *et al*. [27], 11.9% of the *P. aeruginosa* isolates were resistant to ceftazidime. In Belgium and Jamaica a lower level of resistance was found whereas the level was higher in Lagos [27, 33]. *P. aeruginosa* had lower level of resistance against ceftriaxone (a third generation cephalosporin) in this study. This comparatively lower rate of resistance may be due to the relative high cost of the drug and the poor socio-economic status of majority of the people in this environment. As frequent use of drugs tend to induce selective pressure on multi resistant strains [27].

Gentamycin, an aminoglycoside which was second most effective antibiotic against *P. aeruginosa* in this study was reported to have a poor activity (6.9%) against *P. aeruginosa* in a research conducted by Muller-Premru and Gubina [34]. Streptomycin, another aminoglycoside had a low activity against most of the *P. aeruginosa* isolates recording 65.5% resistant. Plasmid-mediated resistance to streptomycin, gentamycin and amikacin were also identified in a study by Daini and Charles-Onyeaghala [29] at Ibadan, South western Nigeria. The increasing resistance of *Pseudomonas aeruginosa* strains to aminoglycosides is in agreement with previous studies [13, 25, 29, 33].

The quinolones used in this study were found to be effective against *P. aeruginosa* isolates tested with few resistant cases. Levoxin, a quinolone was third effective antibiotic against *P. aeruginosa.* Resistance of *P. aeruginosa* to ciprofloxacin another quinolone was 35.5%, compared to 26.8% in Latin America [35] and 10–32% in Europe [36-38]. *P. aeruginosa* isolates had high resistant against ofloxacin 60.0%. The difference in the resistance patterns to the various quinolones is similar to a study in Turkey where a wide range of resistance status against various quinolones was also recorded [39]. The main mechanism of resistance to fluoroquinolones has been reported to be the decrease in binding of the target quinolones to enzymes as a result of changes in DNA gyrase and or topoisomerase enzymes. Mutations occur in gyr A and par C genes. This is usually against all quinolones. However, resistance due to mutations of gyr B, though less common may not be against all quinolones [39].

An exceptionally high resistance to quinolones was reported by Enabulele *et al*. [40] in Benin, Edo state. This is not the case in the present study. Apart from the findings in Benin, high quinolones resistance have also been reported in *P. aeruginosa* isolated from other parts of Nigeria [41-42]. One of the least resistance in the isolates used in this study was observed in ciprofloxacin. This is contrary to the findings by Nwanze *et al*. [43] when they studied UTI in Igbinedion University Teaching Hospital, Delta State. They reported that ofloxacin was the most effective followed by sparfloxacin, pefloxacin then ciprofloxacin. Idu and Odjimogho [44] once showed that ciprofloxacin is the most effective quinolone during their study. Other reports from Iran showed 15.4% resistance to the ciprofloxacin [45]; and Greece 31% [46]. This report is in conformity with the result of this study in which ciprofloxacin recorded one of the least resistance (35.5%) to P. aeruginosa isolates from wound infection patients. Similar reduced resistance of P. aeruginosa to ciprofloxacin has been reported in in Jamaica (19.6%) [33], Latin America (28.6%) [35], India (26.22%) [47], Ilorin Nigeria (24.7%) [48] and Kuala Lumpur, Malaysia (11.3%) [49]. This goes to show that regional differences probably play a role in the resistance profiles of bacteria and further justifies the need to undertake antibiotic susceptibility studies on bacterial isolates from different parts of Nigeria on a regular basis.

From the results of this study ceftazidime, may be considered as empirical therapy of first choice for *P. aeruginosa* wound infections in south west, Nigeria followed by gentamycin and levoxin though according to Gilbert *et al.* [50], ceftazidime, gentamycin and ciprofloxacin were second line of antibiotics administered for wound infections at hospitals in Mandeville, Kingston and St. Andrew. The difference in susceptibility or resistance patterns demonstrated in different geographic locations may be attributable to factor like exposure to antibiotics.

Tetracycline which is traditionally used in this part of the world against wound infections had a low activity (29.1%) against *P. aeruginosa*. This might be as a result of misuse or overuse of this antibiotic over many decades.

The plasmid analyses revealed that there were detectable plasmids in eight (36.4%) out the 22 *P. aeruginosa* isolates, while 14 (63.6%) had no plasmid bands. All the isolates that had plasmids were resistant to amoxicillin, ampicillin, cloxacillin, cotrimoxazole, erythromycin and tetracycline. The sizes of the plasmids among *P. aeruginosa* isolates ranged from 662bp to 830bp. Plasmid mediated resistance to various antimicrobial drugs have been demonstrated by various workers [13, 26-28]. *Pseudomonas aeruginosa* isolate P84 was resistant to gentamycin. In a study at Lagos University Teaching Hospital (LUTH), Lagos, Nigeria, resistance to gentamycin, was attributed to transferable plasmids [51]. In another study done in Greece, plasmids isolated from multi-resistant *P. aeruginosa* strains were found to encode high level resistance to gentamycin [52]. In a few cases of outbreaks in Korea, Japan and Turkey, plasmids encoding potent β-lactamases together with aminoglycoside-modifying enzymes were disseminated among *P. aeruginosa* strains rendering control even more difficult [9].

From this study, 100.0% of the isolates were resistant to the 15 antibiotics used in this study; this is quite worrisome due to the fact that if the majority was resistant to 15 antibiotics constant antibiotic screening must be done before drugs are prescribed for *Pseudomonas aeruginosa* infections in our environment. This result is similar to that of Smith *et al*. [15] with 60.0% and Tassios *et al*. [46] with 52.0% of their isolates being multidrug resistant.

A study of *P. aeruginosa* isolated from urine of students at Ahmadu Bello University, Zaria showed uniform susceptibility to ciprofloxacin [53]. In other part of Nigeria, several studies have reported on the sensitivity of *Pseudomonas* isolates to fluoroquinolones (>60%) [15, 25, 48, 54], while there was varying sensitivities to the cephalosporins cefuroxime (76.6%) and ceftazidime (50.7%) [48]. In another study by Yoon *et al*. [55], 56% of Korean *Pseudomonas aeruginosa* isolates were multidrug resistant (MDR) out of which 44% showed resistance to five or more antibiotics.

Pseudomonas resistant to third generation cephalosporins is real threat. In fact, the irrational and inappropriate use of antibiotics is responsible for the development of resistance of Pseudomonas species to antibiotic monotherapy [47]. Periodic susceptibility testing should be carried out over a period of two to three years to detect the resistance trends [47]. Also, a rational strategy on the limited and prudent use of antipseudomonal agents is urgently required. The incidence of *Pseudomonas aeruginosa* in postoperative wound infection is becoming more serious in developing countries because of relaxation in general hygienic measures, mass production of low quality antiseptic and medicinal solutions for treatment, difficulties in proper definition of the responsibility among the hospital staff [47].

In conclusion, there is an alarming increase of infections caused by antibiotic-resistant bacteria. This study has highlighted diverse plasmid profiles and wide spread antimicrobial resistance patterns of some clinical strains of *Pseudomonas* a*eruginosa* from Nigeria. Therefore the rational use of antimicrobials must be a priority. Public health policy on appropriate prescribing and use of antibiotics must be instituted and affected.

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