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Original Research Article

MDR Pseudomonas aeruginosa isolate from wound infection in Rajshahi Medical College Hospital

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Abstract: ***Background*:** Emergence of resistant bacteria causing nosocomial infections increases the morbidity and mortality. Antibiotic resistance is a major problem in treating infections in hospitals. During the past decade, infecting bacteria that are resistant to several available antibiotics have emerged. The worldwide spread of multi drug resistant bacteria specially Pseudomonas aeruginosa is a predominant isolate which is usually multi drug resistant. ***Objective:*** To identify Antibiotic resistance pattern of Pseudomonas aeruginosa isolated from wound infection. ***Methods:*** A descriptive type of cross-sectional study on wound infection was carried out in the Department of Microbiology of Rajshahi Medical College, Rajshahi during the period from July 2014 to June 2015. A total of 150 wound swabs were collected from patients admitted in surgery and its allied branches and cultured on appropriate bacteriological culture media. ***Results:*** Culture had yielded growth in 131(87.33%) cases and Pseudomonas aeruginosa was 27(18%), Staphylococcus aureus was 22(14.66%), Escherichia coli was 56(37.33%), Proteus spp was 19(12.67%), Klebsiella spp. was 7(4.67%) respectively. Antibiogram was tested on Pseudomonas aeruginosa with 7 different groups of antibiotics and found 4(14.81%) were resistant to 3 groups of drugs, 2(7.41%) were resistant to 4 groups, 5(18.52%) were resistant to 5 groups, 10(37.04%) were resistant to 6 groups and 6(22.22%) were resistant to 7 groups. A total of 27(18%) isolates were resistant to 3-7 groups of antibiotics. ***Conclusion:*** Pseudomonas aeruginosa is still a predominant isolate next to E. coli for wound infections. 27(18%) isolates were resistant to 3-7 groups of commonly used antibiotics. Recommendation: All wound infections should be treated only after performing antibiogram with adequate dose and duration of antibiotics.

Keywords: Wound Infection, Pseudomonas Aeruginosa, Multidrug Resistance, Hospital Patients.

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Introduction

Wound infections are one of the most common infections in hospital which include skin and soft tissue infection. Skin is the largest organ of human body and plays a vital role to protect the underlying tissue against colonization and invasion by bacteria. But loss of integrity of the skin provides a moist, warm and nutritive environment for bacterial colonization, proliferation and causing wound infection.[1] Wound infections may be hospital acquired (nosocomial) or community acquired. Hospital acquired infections is about 5% to 34% in both developed and developing countries and it may cause by both aerobic and anaerobic bacteria and fungus also.[2] Clinically WI may be traumatic, burn, surgical infection and bed sore due to diabetic. Whatever is the nature of infection, Pseudomonas is considered as a major hospital problem. This bacterium is frequently found in the hospital utility solutions, tap water, sink, mops,

detergent, respiratory and physiotherapy equipment's etc.[3] Hospital personnel may transmit these bacteria from patient to patient while handling patient. It also causes septicemia, urinary tract, respiratory tract and great variety of systemic infections and reported incidences of nosocomial pneumonia was 16%, urinary tract infection was 12%, wound infection was 17-26% and septicemia were 10%.[4] Pseudomonas is an opportunist pathogen with resistance to β-lactams, quinolones, chloramphenicol and tetracyclines. It also develops resistance due to very low permeability of bacterial cell wall which is depends up to the production of cephalosporinase, active efflux and poor affinity for the target.[5]

It develops resistant due to mutation in chromosomal genes, acquisition of resistant genes from same or different species of bacteria via plasmids or transposons by conjugation and transduction.[6] All these mechanisms make Pseudomonas most difficult bacteria to treat. A large number of acquired resistance genes for β-lactamases, extended-spectrum β-lactamases and metallo-β-lactamases have been detected in Pseudomonas.[7] In recent years increase prevalence of multidrug resistance in P. aeruginosa has been noticed. A limited number of antibacterial agents such as ticarcillin, piperacillin, cephalosporins, carbapenems and fluoroquinolones are effective against Pseudomonas aeruginosa. Aminoglycosides are also used as a part of combination therapy .[8] So the present study has been carried out to determine pathogens responsible for wound infection and antibacterial resistance pattern of Pseudomonas aeruginosa.

Materials and Methods

This study was conducted in the Department of Microbiology of Rajshahi Medical College, Rajshahi during the period from July 2014 to June 2015. A total of 150 wound swabs were collected from patients admitted in Surgery and its allied branches. The samples were cultured on blood agar, nutrient agar and MacConkey's agar. Pseudomonas aeruginosa was identified by its colony morphology, microscopy, motility, pigment production, fruity odour and oxidase positivity. Antimicrobial susceptibility test was performed on Mueller-Hinton agar media with commercially available antibiotic discs by disc diffusion method. Antibiotic discs were meropenem (10µg), ciprofloxacin (5µg), ceftriaxone (30µg), aztreonam (30µg), gentamicin (10µg), tigecycline (15µg) and ticarcillin (75µg). The result was reported as sensitive and resistant according to CLSI, 2012 recommendation. The strains which showed resistance to more than 3 drugs of different mode of actions were considered as multidrug resistant (MDR).

Results

In this study 27(18%) Pseudomonas aeruginosa were isolated from 150 wound swabs. Among 27 isolates ceftriaxone was resistant to 25(92.59%), meropenem was 9(33.33%), gentamicin was 22(81.48%), aztreonam was 18(66.67%), tigecycline was 25(92.59%), ciprofloxacine was 22(81.48%) and ticarcillin was resistant to 26(96.3%) isolates (Table 1). Among 27 Pseudomonas aeruginosa, 4(14.81%) were resistant to 3 groups of drugs, 2(7.41%) were 4 groups, 5(18.52%) were 5 groups, 10(37.04%) were 6 groups and 6(22.22%) were resistant to 7 groups of drugs (Table 2).

Table 1: Antibacterial Resistance of Pseudomonas Aeruginosa. (N=27)

	Isolates number of Pseudomonas aeruginosa																										
Antibacterial groups with specific drug.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
Cephalosporins: Ceftriaxone(30µg)	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	S	R	R
Carbapenem:	S	S	S	S	S	S	S	S	R	S	S	S	S	R	R	R	R	R	R	R	S	S	S	S	R	S	S

Meropenem(10µg)																										
Aminoglycoside: Gentamicin (10µg)	R	R	R	R	S	S	S	S	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R	R
Monobactam: Aztreonam (30µg)	R	R	R	R	S	S	R	S	S	R	R	R	S	R	S	R	R	R	R	R	R	S	R	R	S	S	R
Tetracycline: Tigecycline (15µg)	R	R	R	R	R	R	S	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R	R
Fluoroquinolone: Ciprofloxacin(5µg)	R	R	R	R	R	S	S	S	S	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R
Penicillin Ticarcillin (75µg)	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	S	R	R
No. of resistant.	6	6	6	6	4	3	3	3	5	6	6	6	5	7	6	7	7	7	7	7	6	5	5	4	3	5	6

Note: R= Resistant; S= Sensitive

Table 2: Multidrug Resistance Patterns of Pseudomonas Aeruginosa. (N= 27)

	No. of antibacterial drugs in group.				
Resistant Pseudomonas aeruginosa.	3	4	5	6	7
	4 (14.81)	2 (7.41)	5 (18.52)	10 (37.04)	6 (22.22)

Note: N= number
Figures within parenthesis indicate percentages.

Discussion

In this study seven groups of antibiotics were studied for a sensitivity test. The groups were cephalosporin, carbapenem, aminoglycoside, monobactam, tetracycline, fluoroquinolone and penicillin. Among cephalosporins, ceftriaxone was 92.59% resistant which is similar with the study of Rostamzadeh *et al.* in Iran and Mahmoud *et al*. in Egypt where ceftriaxone resistant was 94.37% and 87.7%.[9, 10] But dissimilarity was found with the study of Garba *et al.* and Mohammed *et al.* both were in Nigeria and showed ceftriaxone resistant was 45.4% and 46%.[11, 12] Among the carbapenems, meropenem was 33.33% resistant which is similar with the study of Joseph *et al*. in India and Khan *et al.* in Pakistan where meropenem resistant was 34.8% and 30.4%.[13, 14] The dissimilarity noted by Yasemin *et al.* in Turkey and Biswal *et al.* in India where rates were 19% and 13.79%. [15, 16] Gentamicin was 81.48% resistant which is similar to the study of Rajput *et al.* and Biswal *et al.* both were in India where gentamicin resistant was 81% and 81.03%. [16, 17] Dissimilarity with our study was reported by Shah *et al.* in Pakistan and Yasemin *et al.* in Turkey where resistant was 35.3% and 36%.[15, 18]

Aztreonam was 66.67% resistant which is nearly similar to the study of Nazli *et al*. in Turkey where aztreonam resistant was 56.7% and dissimilar with the study of Khan *et al.* in Nepal and Mahmoud *et al*. in Egypt where resistant was 31.96% and 82.5%.[10, 19, 20] Tetracycline was 92.59% resistant which is similar

with the study of Smith *et al.* in Nigeria and Mohiuddin *et al*. in Dhaka, Bangladesh where resistant was 95% and 91.17%.[21, 22] But our study is dissimilar with the study of Akingbade *et al.* in Nigeria and Masood *et al*., in Iran where resistant rates were 70.9% and 72%.[23, 24] Ciprofloxacin was 81.48% resistant which is nearly similar to the study of Mohiuddin *et al*., in Dhaka, Bangladesh and Khan *et al.* in Pakistan where ciprofloxacin resistant were 92% and 75%.[14, 22] But dissimilarity with our study was reported by Mahmoud *et al.* in Egypt and Golshani *et al.* in Iran were 56.1% & 58%.[10, 25] Ticarcillin was 96.3% resistant which is similar to the study of Shahini *et al.* and Ranjbar *et al*., both in Iran where resistant rates were 100% and 93%.[26, 27] But dissimilarity was reported by Sarwat *et al*. in India & Masood *et al*., in Iran was 58.46% and 5%.[24, 28]

The resistant pattern of Pseudomonas aeruginosa in our study is different from the studies of others. It may be due to the random use of 3rd generation cephalosporins and carbapenem without doing culture and sensitivity which lead to the emergence of resistance and their dissemination throughout the hospital. This dissemination is due to inadequate sanitation of hospital, improper use of antibiotics, inadequate antibiogram of empirical antibiotics, inadequate dose and duration, may be insufficient ingredients as mentioned by the pharmaceutical company and inaccuracy of culture and sensitivity test. In our study 14.81% Pseudomonas aeruginosa was resistant to 3 antibiotics which is similar to the study of Gobedo *et al*. in Ethiopia where they found 14.9%.[29] Dissimilarity with our study was reported by Biswal *et al.* in India where resistant isolates were 10.34%.[16] 7.41% isolates were resistant to 4 antibiotics which are similar to the study of Yakha *et al*. in Nepal and Odumosu *et al.* in Nigeria where resistant isolates of both were 6.45%.[30, 31] Dissimilarity was reported by Biswal *et al.* in India were 3.45%.[16]

In this study 18.52% isolates were resistant to 5 antibiotics which are similar to the study of Mehdi *et al.* in Iran and Yakha *et al.* in Nepal where resistant isolates were 17.8% and 19.35%.[30, 32] Dissimilarity was reported by Gobedo *et al*. in Ethiopia were 4.1%. In this study 37.04% isolates were resistant to 6 antibiotics which are similar to the study of Mehdi *et al.* in Iran were 38.4%.[29, 32] But dissimilarity was reported by Odumosu *et al*. in Nigeria were 9.68%.[31] In our study 22.22% isolates were resistant to 7 antibiotics which are dissimilar with the study of Gobedo *et al.* in Ethiopia where resistant isolates were 5.4%.[29] The dissimilarities of the multidrug (3-7 drugs) resistant isolates may be due to use of antibiotics in our study is different from others study, different therapeutic dose and route; patients may have different pH in their stomach which may differ the activity of orally administered drugs like ciprofloxacin; food can interfere the absorption of drug e.g. tetracycline; milk, antacid, sucralfate and iron salt may reduce the absorption of certain drugs e.g. tetracycline, fluoroquinolone etc. Dissimilarities may also be due to achlorhydia, partial gastrectomy, tropical sprue where absorption of drug reduce and cannot reach at optimum serum concentration. In oral administration as only 20-40% drug reaches the systemic circulation while 100% in parenteral administration, metabolism may also alter the efficacy and half-life of drug. Besides these oral formulation of a drug from different manufacturers or different batches from the same manufacturer with same amount of drug may not yield the same blood levels. Mutation may occur in bacteria if optimum blood level is not attained by orally administered drug that also causes antibiotic resistance.

Conclusions

It can be concluded that Pseudomonas is a classic opportunist pathogen with innate resistance to many antibiotics due to mutation in their chromosomal genes, acquisition of resistant genes from same or different species of bacteria via plasmids and transposons by conjugation and genome based resistant genes by transduction. Intrinsic and extrinsic (acquired) antibiotic resistance makes Pseudomonas one of the most difficult bacteria to treat. From this study, it has found that Pseudomonas aeruginosa isolates are resistant to commonly used antibiotics and their resistance to antimicrobials gradually increases day by day. Therefore, the rational use of antibiotics must be a priority. Public health policy on appropriate prescribing and antibiotics should be used only after performing antibiogram with adequate dose and duration.

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