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Mrk D and fim H Biofilm Genes Detection and Antibiotic Susceptibility: A Study on Uropathogenic Biofilm Producing Klebsiella pneumoniae in Sana'a City, Yemen

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Article

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Abstract

Background

Urinary tract infections (UTIs) caused by antibiotic-resistant strains of *Klebsiella pneumoniae* (*K. pneumoniae*) are one of the most important causes of morbidity and health care spending affecting people in Yemen, including young women, children, and the elderly. Biofilm formation has become a major factor among those uropathogenic strains.

Objectives

This study aimed to investigate the biofilm-forming ability of *K. pneumoniae* isolated from patients with UTIs, to determine the antibiotic susceptibility of those biofilm producers, and to assess the association between fimbrial virulence genes **(***fimH* and *mrkD***)** and biofilm strength.

Methods

The standard procedures for identifying and isolating bacteria from UTIs were used. The ability of the isolated *K. pneumoniae* to form biofilms was examined using tissue culture plates. The Kirby-Bauer disc diffusion method was used to determine the antibiogram susceptibility pattern of uropathogens. The fimbriae virulence genes (*fimH* and *mrkD*) for the biofilm-producing *K. pneumoniae* were found using conventional PCR.

Results

From UTI patients, 79(32.8%) of *K. pneumoniae* were isolated. 39 UTI patients (49.4%) had *K. pneumoniae* that did not produce a biofilm, although 40 (50.6%) of the bacteria are capable of doing so. Among those biofilm-forming strains, 19 (47.7%) were able to produce strong biofilms, while 21 (52.5%) produced moderate biofilms. PDR (5%), on the other hand, had the lowest percentage of antibiotic resistance, whereas XDR (47.5%) and MDR (32.5%) had the greatest percentages. The *mrkD* gene was detected in 40 (100%) of the biofilm-forming *K. pneumoniae*, and *fimH* had a significant association with strong biofilm ($p = 0.031$).

Conclusion

The presence of fimbriae virulence genes (*mrkD* and *fimH*) was significantly correlated with multi-drug resistance (MRD) biofilm-producing *K. pneumoniae* isolated from UTI patients.

Introduction

One of the most prevalent clinical Gram-negative bacteria, *K. pneumoniae*, can cause serious infections, including wound infections, pneumonia, and urinary tract infections [1, 2, 3]. Due to the high death rate from *K. pneumoniae* infections, particularly in children and patients with impaired immune systems, a great deal of research has focused on the development of antibiotic resistance [4]. Indeed, it has been documented that *K. pneumoniae* is a common cause of infections in people who have indwelling urinary catheters [5, 6, 7]. Multiple processes allow this bacterium to become resistant to antibiotics, leading to the development of multidrug-resistant (MDR) strains that pose a severe threat to patient safety and medical center architecture. The production of biofilms is a significant element in the initiation and propagation of infection [8].

The ability of bacteria to live in unfavorable environments is known as biofilm development [9]. This biofilm shields the bacteria from antibiotics and the human immune response, making the bacteria within highly resistant to antibiotic treatments [10]. Multidrug-resistant organisms (MDRO) are able to spread more easily in biofilms because they offer an environment that is resistant to antibiotic penetration and allows for the horizontal transfer of virulence genes [11]. Biofilm-forming bacteria are responsible for about 80% of urinary tract infections (UTIs), which is a severe medical concern. In addition, biofilm contributes significantly to recurring UTIs and catheter-associated UTIs [12]. Since biofilm formation by pathogenic bacteria may be a virulence factor in individual attacks, it is currently of interest. This emphasizes the need for ongoing assessment of pathogen development [13].

The virulence elements primarily responsible for *K. pneumoniae's* capacity to form a biofilm include the type 1 or type 3 fimbriae, the capsule, and the lipopolysaccharides. It has been shown that type 3 fimbriae are the main appendages that mediate biofilm formation on abiotic surfaces and adhesion to endothelium and bladder epithelial cell lines [14]. Specifically, the type 3 fimbrial protein *mrkA* promotes *K. pneumoniae* growth on abiotic surfaces, while the type 3 fimbrial adhesin *mrkD* is necessary for development on surfaces covered with human extracellular matrix [15].

After *E. coli, K. pneumoniae* is the second pathogen that causes UTIs in Yemen [6, 7, 16, 17]. Although UTIs pose a significant threat to Yemeni society, little is known about the expression of virulence genes in biofilm-forming *K. pneumoniae* UTIs and how these genes relate to antibiotic resistance. To ascertain the antibiotic susceptibility of *K. pneumoniae* isolates from UTI patients in Sana'a City, our study aimed to investigate the biofilm-forming capacity of *K. pneumoniae* isolated from patients with UTIs, to determine the antimicrobial susceptibility patterns of biofilm-producing isolates, and to detect the prevalence of common fimbrial virulence genes (*fimH* and *mrkD*), as well as to evaluate the association between the presence of these genes and the strength of biofilm formation.

Subjects and Methods

This descriptive cross-sectional study was conducted at Al-48 Modern General Hospital and the University of Science and Technology Hospital in Sana'a City. The laboratory works were performed at the Department of Microbiology and Molecular Biology at the National Centre of Public Health

Laboratories (NCPHL), Sana'a city, Yemen, during a period of one year, starting in February 2024 and ending in February 2025. About 10 to 20 ml of midstream urine was collected in a sterile screw-capped wide-mouth container from all participating UTI patients.

Isolation and identification of K. pneumoniae

The urine sample with leukocyte counts > 10 WBCs/HPF was cultured using a standard calibrated loop to contain 0.01 ml of urine on cysteine-lactose-electrolyte-deficient (CLED) agar media. Inoculated plates were incubated at 37°C aerobically for 18–24 hours. After incubation, the plates were examined, and growth characteristics were noted. Samples were usually inoculated before cell content analysis to reduce the risk of sample contamination. Colonies were counted on the inoculated medium and multiplied by the loop volume to determine the bacterial count (significant growth > 10^5 CFU/mL). Bacteria were identified using the automated Vitek2 system, conducted according to the manufacturer's instructions (BioMérieux, France).

Biofilm detection

The isolated bacteria were tested for biofilm production on a tissue culture plate. The classification of bacterial adherence by the tissue culture plate method into three categories: average optical densities (OD) value < 0.120 (non-adherent), 0.120–0.240 (Moderate), and > 0.240 (strong) biofilm formation (Fig. 1).

Determination of the antibiogram susceptibility pattern

The antibiogram susceptibility pattern of uropathogens to different antibacterial agents was determined *in vitro* by employing a modified disk diffusion method described by Kirby-Bauer [18]. The procedure involved measuring the diameter of the zone of inhibition that results from the diffusion of the antibiotic agent into the medium surrounding the disc. The interpretation of the obtained results was based on the Clinical Laboratory Standards Institute (CLSI) guidelines [19].

Identification of K. pneumonia Genes

The conventional polymerase chain reaction (PCR) was used to detect the sequences of virulence genes (*mrkD* and *fimH*) for the biofilm-forming K. pneumoniae using specific forward (F) and reverse (R) primers that were previously used in a study conducted by Shah *et al*. [20] (Table 1).

Table 1
Primers used to detect the sequences of the mrkD and *fimH* gene this study

Gene	Primer Sequence (5–3)	Expected size (bp)
*fim*H	F: 5´- ATG AAC GCC TGG TCC TTT GC -3´	688
	R:5´-GCT GAA CGC CTA TCC CCT GC -3´	
*mrk*D	F: 5´- CCA CCA ACT ATT CCC TCG AA -3´	240
	R: 5´- ATG GAA CCC ACA TCG ACA TT -3´	

Statistical analysis

The data was analyzed using SPSS Version 26. The qualitative variables were presented as frequencies and percentages. Associations between the positive fimbriae virulence genes and the biofilm-producing capacity of *K. pneumoniae* were tested using chi-square tests, where a p-value < 0.05 was considered a significant result. The relative risk with a 95% confidence interval was also determined.

Results

A total of 241 urine samples from UTI patients were collected in this study. The rate of *K. pneumoniae* was 79 (32.8%) isolates in UTI cases **(**Fig. 2**).** Among 79 isolates, 40 (50.6%) of *K. pneumoniae* can form a biofilm, while non-biofilm-producing *K. pneumonia*e were found in 39 (49.4%) of UTI patients. The biofilm-producing *K. pneumoniae* gives a strong biofilm formation in 19 (47.7%) and moderate in 21 (52.5%) of isolates, while no cases show weak biofilm **(**Fig. 3A **and B).**

Regarding the antibiogram susceptibility pattern among the isolated biofilm producing K. pneumoniae, the highest percentage of antibiotic resistance was for XDR (47.5%), followed by MDR (32.5%), while the lowest percentage of antibiotic resistance was for PDR (5%) **(Fig. 4).** However, the sensitivity to antibiotics among the biofilm-producing *K. pneumoniae* was only 6 (15%).

In this study, all 40 isolates (100%) of biofilm-producing *K. pneumoniae* appeared to express the *mrk*D gene; 19 (47.5%) and 21 (52.5%) were among the strong and moderate biofilm-producing *K. pneumoniae*, respectively. While the *fim*H gene was found in 35of 40 (87.5%) isolates, 19 (54.3%) and 16 (45.7%) of high and moderate biofilm-producing *K. pneumonia*, respectively. The statistically significant association with a significant risk of more than two times between positive *fim*H gene and strong biofilm-formation (RR: 2.19; CI: 1.5–3.1; p = 0031) (Table2).

MDR: multidrug-resistant, XDR: extensively drug-resistant, PDR: pan-drug-resistant

Figure 4: The antibiogram susceptibility pattern among the isolated biofilm-producing *K. pneumoniae*

Table 2
The prevalence of fimH and mrkD genes among the isolated biofilm-producing *K. pneumoniae* and their associations with biofilm strength.

Fimbriae virulence genes	**Biofilm producer**				**PR (CI 95%)**	***p*-value**
	Moderate		**Strong**			
No.	**%**	**No.**	**%**			
Fim H (n = 35)	16	45.7	19	54.3	2.19(1.5–3.1)	0.031
Mrk D (n = 40)	21	52.5	19	47.5	UD	UD
CI: Confidence interval; *PR* Prevalence ratio; UD: Undefined						

Discussion

K. pneumonia has the capability of biofilm formation, which protects it from host immune system responses and antibiotics [20] and promotes its persistence on the epithelial tissues and external surfaces of indwelling devices. This study showed that 50.6% of *K. pneumoniae* isolates were capable of forming biofilm, which gives strong biofilm production in 47.7% and moderate biofilm production in 52.5% of isolates. Several global studies recorded higher than our findings. Makhrmash et al. reported that all isolated *K. pneumonia* 100% were biofilm producers, with 57% of isolates producing strong biofilm [21]. Hamam *et al*. showed that the majority (81%) of the isolates in their studies had the capacity to form biofilm [22]. Another related study conducted in Iran showed that 79.6% of *K. pneumoniae* isolated from urine specimens were biofilm producers, where 23% of them could produce strong biofilm [23]. Research from Nigeria revealed that 68.18% of isolates were biofilm producers [24]. The formation of biofilm protects *K. pneumoniae* against the host immune responses, the action of antibiotics, and enhances its persistence [25]. The discrepancy in the biofilm formation demonstration between our results and the previous literature may be due to different bacterial strains, resistance profiles, genes, variations in the sites of isolates, and the culture conditions and detection method [21, 26].

Our findings showed that XDR was found in 22 (55%), followed by MDR, which represented 15 (37.5%), and PDR, which counted 2 (5%) of all biofilm-producing *K. pneumoniae*. However, the sensitivity to antibiotics among the biofilm-producing *K. pneumoniae* was only 6 (15%). This result is similar to reports from Pakistan, which showed all isolates of *K. pneumoniae* were highly resistant to the majority of antibiotics [24]. Two studies conducted elsewhere in which antibiotic resistance was significantly high among biofilm-producing *K. pneumoniae* [27, 28]. In general, this may be because bacterial biofilms are often associated with long-term persistence of organisms in various environments, decreased bacterial growth rate in a biofilm, expression of resistance genes, and restricted penetration of antibiotics into the biofilm. Furthermore, the proximity of cells within a biofilm can facilitate plasmid exchange and hence enhance the spread of antimicrobial resistance [29].

Regarding the fimbriae virulence genes, this data showed a significant association between the positive *fim*H gene and the strong biofilm-formation (p = 0.031). Three studies were in agreement with our finding [21, 22, 30, 31]. The FimH has a significant role in pathogenicity and resistance among strains of K. *pneumoniae* due to enhancing biofilm formation [32]. Meanwhile, the *mrkD* gene was discovered to be present in all biofilm-producing *K. pneumoniae* isolated from UTIs 100% in this investigation, which determined the virulence genes (*mrkD* and *fimH*) among biofilm-producing *K. pneumoniae*. This finding aligned with two studies conducted in Iraq that demonstrated that all biofilm-forming *K. pneumoniae* isolates from UTIs 100% tested positive for the *mrkD* genes [14, 33]. Numerous investigations have demonstrated that type 3 fimbriae (*mrkD* gene) appear to play important roles in *K. pneumoniae* biofilm formation, which is consistent with our results [15, 34]. The observation that mutant *K. pneumoniae* strains lacking this gene cluster produced noticeably less biofilm in the presence of lung surfactant, with cholesterol and phosphatidylcholine identified as surfactant components promoting type III fimbriae expression [35].

Among the limitations of the present study include; the study design, a cross-sectional study in which data was collected at one point of time and the causative are not detected and for financial issues such as the tested panel of antibiotic was not including all antibiotics used in treatment of urinary tract isolates and detection of only mrkD and fimH as two biofilm-associated genes without involving other complex genetic determinants may affect the findings.

Conclusion

The presence of fimbriae virulence genes (mrkD and fimH) was significantly correlated with multidrug-resistant biofilm-producing *K. pneumoniae* isolated from UTI patients.

Declarations

Ethics approval and consent form

Ethical approval:

The ethical approval of this study was obtained from the Medical Ethical Committee of the Faculty of Medicine and Health Science, Sana'a University; No. (287). Ethical approval was obtained in accordance with the standards outlined in the Helsinki Declaration. Written informed consent forms were obtained from all participated UTIs patients before any research procedure was performed.

Competing interests

The authors declare that there is no conflict of interest.

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Author Contribution

HAA supervision and revision, and editing of the manuscript. SMMD collected data and samples from all patients and performed the laboratory procedures for the samples. DAE and KAA wrote of introduction and methodology. HAA and ANMG analyzed the data, prepared tables and figures, and discussed the results. All authors arranged the referencing style, read, and made the final revision of the manuscript. All authors have read and agreed to the published version of the manuscript.

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Data Availability

The data will be available to anyone upon request from the corresponding author.

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Figures

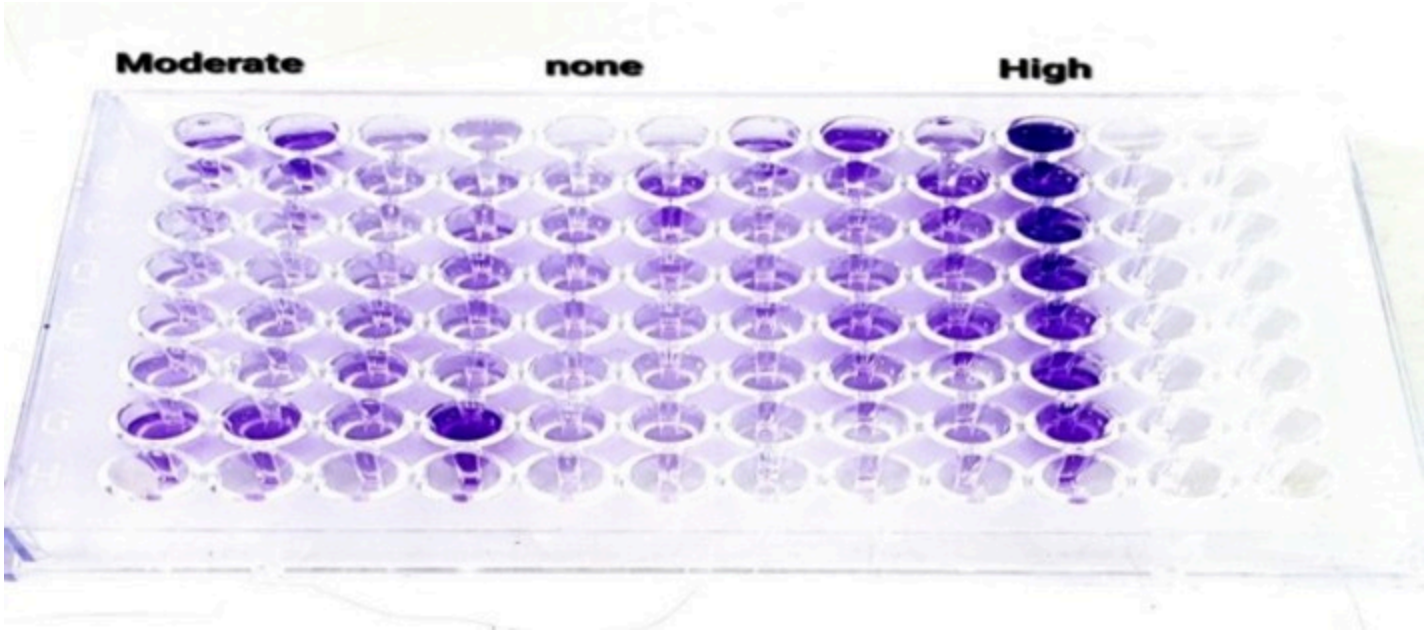


Figure 1

Tissue culture plate

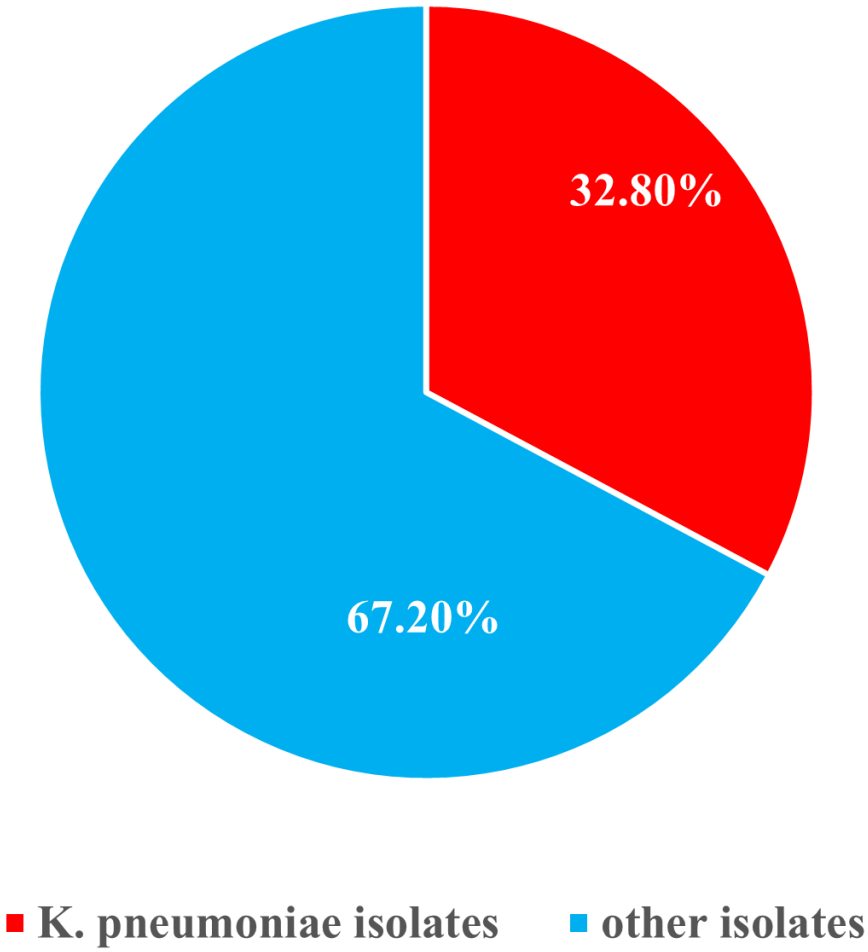


Figure 2

The rate of *K. pneumoniae* isolates from UTI cases (n=241).

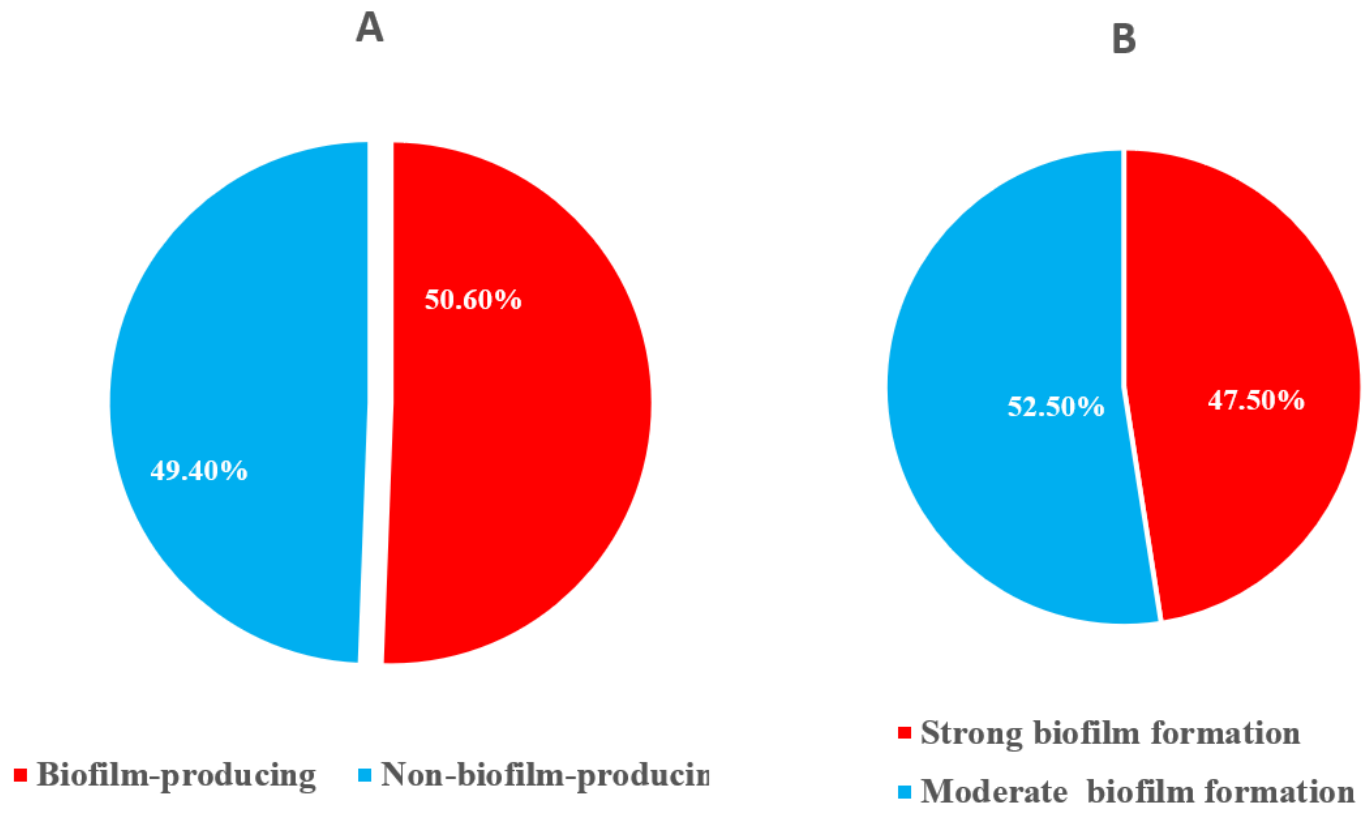


Figure 3

A) The rate of biofilm-producing K. pneumoniae, B) The strength of biofilm among biofilm-producing *K. pneumoniae*

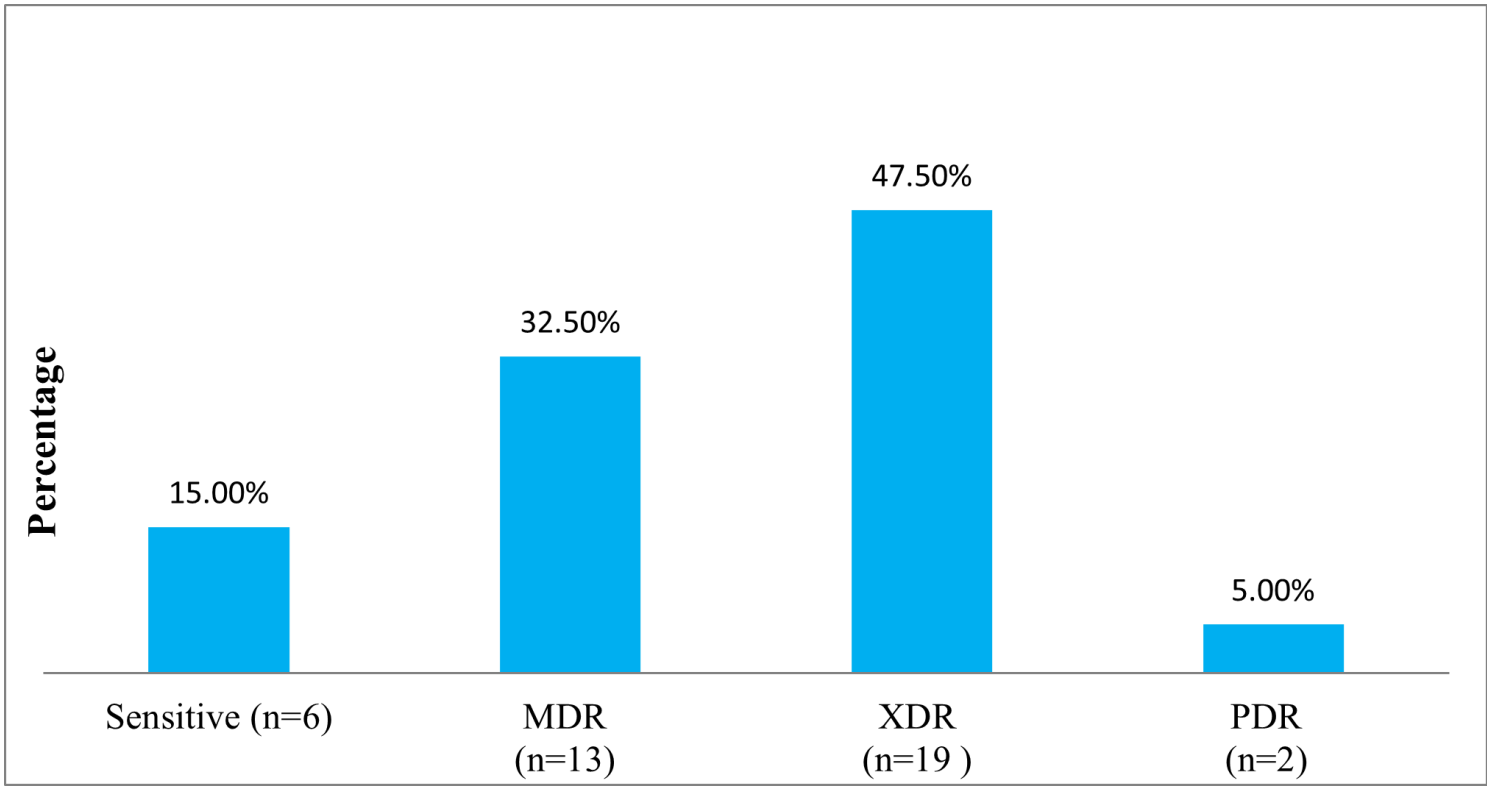


Figure 4

The antibiogram susceptibility pattern among the isolated biofilm-producing *K. pneumoniae*