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Prevalence and Occurrence of Type 1 Fimbriae in *Klebsiella pneumoniae*

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ABSTRACT

Background: *Klebsiella pneumoniae* causes both community acquired and nosocomial infections. The various virulence factors have been well characterised in *K. pneumoniae* includes: capsule, lipopolysaccharides, siderophores and fimbriae. *FimH* 1, encoding for fimbriae and mediate adhesion.
Aim and objective: The aim of the study was to determine drug susceptibility and the prevalence of *FimH* 1 genes among clinical isolates of *K. pneumoniae.*
Materials and methods: A total of 200 isolates collected over a period of one year, were included in this study. The source of the isolates were urine (n=74), respiratory (n=73), exudates (n=50) and blood (n=3). For all the isolates antimicrobial susceptibility testing by disc diffusion was done. Polymerase chain reaction was performed for the detection of *FimH* 1 gene.
Results: The susceptibility of the study isolates to different classes of antimicrobial agent was: meropenem (75%), amikacin (69%), piperacillin/tazobactam (67.5%), ciprofloxacin (59%), and cefotaxime (53.5%). *FimH-1* gene was detected in 55% of the total isolates.
Conclusion: *FimH* 1 was not a major mediator associated with adherence in this study. Detection of virulence gene such as type 1 fimbriae will help to understand their occurrence in different strains of *K. pneumoniae* and how they function in different host environments. Most of the isolates were resistant to third generation of cephalosporins. Knowing the prevalence of antimicrobial resistance helps to formulate infection control practices and formulating antimicrobial therapy.

Keywords: *Klebsiella pneumoniae*, virulence gene, fimbrial adhesin, type 1 fimbriae, antimicrobial susceptibility, disc diffusion.

INTRODUCTION

Klebsiella pneumoniae (*K.pneumoniae*) is an important opportunistic pathogen that causes urinary tract infections, septicemia or pneumonia, especially in the immunocompromised.[1] There are four major classes of virulence factors that have been well characterised in *K.pneumoniae* includes; capsule, lipopolysaccharides, siderophores and fimbriae. [2] Adherence to host cell is the first step in the infectious process. In *Enterobacteriaceae*, adhesive properties are mediated by different types of

pili or fimbriae. They are non-flagellar filamentous projections on the bacterial surface. [3]

The best investigated of the bacterial adhesins are type 1 fimbriae, they are mannose sensitive hemagglutinins (MSHA) which agglutinate erythrocytes of guinea pig.[4] Type 1 fimbriae are present in many species of *Enterobacteriaceae*, which mediate adhesion to mannose containing structures on host cells and extracellular matrix. [5]

Currently, *K.pneumoniae* is showing resistance to different classes of antibiotics such as beta-lactam group of drugs, fluoroquinolones and aminoglycosides. The increase in resistance to different classes of antibiotics is a worldwide problem, which limiting the choice of therapeutic options for nosocomial infections caused by *K.pneumoniae*.[6]

The aim of the study was to determine drug susceptibility and the prevalence of *FimH* 1 genes among clinical isolates of *K.pneumoniae.*

MATERIALS AND METHODS

Bacterial isolates

The study included 200 clinically significant, consecutive, non-duplicate isolates of *K.pneumoniae*, collected over a period of one year. The source of the isolates were urine(n=74), respiratory secretions(n=73) exudative specimens(n=50), and blood(n=3).

Antimicrobial susceptibility test

Antibiotic susceptibility testing was done by Kirby-Bauer disc diffusion method as per the Clinical and laboratory standards institute guidelines for cefotaxime (30 µg), ceftriaxone (30µg), amikacin(30µg), ciprofloxacin (5µg), piperacillin/tazobactam (100µg/10µg) and meropenem (10µg) (HiMedia laboratories, Mumbai, Maharashtra, India) as per the Clinical and Laboratory Standards Institute guidelines.[7]

Detection of *FimH*-1 gene by Polymerase chain reaction

Template DNA of the isolates was extracted by boiling method.[8] All the isolates were tested for *FimH*-1 gene, using the primers which are previously described.[9] The primers were *FimH*-1 Forward-ATGAACGCCTGGTCCTTTGC and *FimH*-1 Reverse GCTGAACGCCTATCCCCTGC.

Amplicon size of the gene was 688bp.PCR was performed from Sri Balaji Medical College and research Institute, Chennai.

RESULTS

The susceptibility of the study isolates to different classes of antimicrobial agent was: meropenem (75%), amikacin (69%), piperacillin/tazobactam (67.5%), ciprofloxacin (59%), and cefotaxime (53.5%).

FimH-1 gene was detected in 55% of the total isolates [figure 1]. Distribution of *FimH* -1 gene in various clinical specimens is depicted in table 1.

Figure 1: Lane 1-100bp ladder.Lane 2 and 3-positive control and test strain of *FimH*

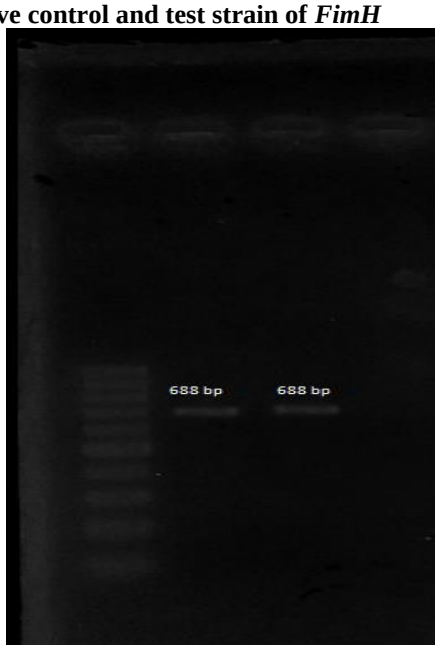

Table 1: Distribution of FimH 1 in various clinical specimens

Specimens (200)	***FimH***
Urine (74)	48.64% (36/74)
Respiratory (73)	53.42% (39/73)
Exudates (50)	66% (33/50)
Blood (3)	66% (2/3)

DISCUSSION

K. pneumoniae is a nosocomial pathogen and a potential community acquired pathogen frequently associated with infections in all age groups, especially in the compromised. They harbours several virulence factors including capsules, lipopolysaccharides and fimbriae.[10] Fimbrial adhesins are protein structures that recognise a wide range of molecular motifs and helps in the adherence of the bacteria to specific tissue surface in the host. [11] Most of the *K.pneumoniae* harbours Fimbrial adhesins.[12]

In the present study, 55% (110/200) isolates expressed *FimH* 1 gene. Study from china reported, 85.5% (53/62) of *K.pneumoniae* harboured this gene.[13] A previous study has detected type 1 fimbriae in 89% of the isolates.[14] In Spain and Iran, the presence of *FimH* gene was 98.43% and 91% respectively. [15,16] Ferreira *et al*. reported *FimH* 1 in 88% of the isolates.[6]

Expression of this gene was high in exudative specimens 66% (33/50) followed by blood 66% (2/3), respiratory 53.42% (39/73) and urine 48.64% (36/74). A study reported, *FimH* was prevalent in sputum. [12] However, in a study with mouse lung infection model reported that expressions of type 1 fimbriae have no role in dissemination of the bacteria from the lungs to blood stream. [5] Presence of this gene in all urinary isolates was reported by Aljanaby and Alhasani and El Fertas-Aissani *et al*. [9, 17] Occurence of this gene from blood and wound isolates reported previously. [18]

In this study, majority of the *K.pneumoniae* (46.5%) showed resistance to third generation of cephalosporins. High prevalence of third generation cephalosporin resistance was similar to previous studies. [19,20 ,21]

CONCLUSION

FimH 1 was not a major mediator associated with adherence in this study. Detection of virulence gene such as type 1 fimbriae will help to understand their occurrence in different strains of *K. pneumoniae* and how they function in different host environments. This will also helpful for the development of new molecular detection assays and therapeutic pathways. Awareness regarding the resistance profile prevalent in hospitals helps to implement infection control practices and formulating antimicrobial therapy.

Declaration by Authors

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