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Original Article

Internal Medicine Section

Predisposing Factors of *Pseudomonas* Pneumonia: A Hospital Based Cross-sectional Study

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ABSTRACT

Introduction: *Pseudomonas aeruginosa* (*P. aeruginosa*) is a usual causative organism of both hospital-acquired pneumonia and community-acquired pneumonia. Although, it is less commonly reported in otherwise healthy hosts, most patients have an identifiable risk factor for disease. These risk factors include those with structural lung abnormalities like bronchiectasis and those with a compromised immune system. With the emergence of multidrug resistant strains of *P. aeruginosa*, the severity of infection and mortality associated with pneumonia has increased drastically. The insight into the risk factors of *Pseudomonas* pneumomia helps to suspect and treat the condition at the earliest and thereby reduce the mortality as well as the duration of hospital stay.

Aim: To determine various risk factors associated with *P. aeruginosa* pneumonia.

Materials and Methods: An observational cross-sectional study was conducted from November 2018 to November 2020 at Jawaharlal Nehru Medical College, Aligarh, Uttar Pradesh, Inida, after obtaining Institutional Ethics Committee (IEC) approval. A total of 89 patients with diagnosis of *Pseudomonas* pneumonia, defined as the presence of signs and symptoms of pneumonia along with sputum or tracheal culture positive for *P. aeruginosa* were enrolled in the study. These patients were thoroughly assessed by clinical history, physical examination and relevant investigations to detect any risk factors like age, diabetes, smoking, Chronic Obstructive Pulmonary Disease (COPD), chronic steroid use and prior hospital admission. Data analysis was done using Statistical Package for Social Sciences (SPSS version 22.0). Categorical variables were compared with the Chi-square test.

Results: Out of 89 patients with *Pseudomonas* pneumonia, 77.5% were above 40 years of age, 37.08% were smokers, 25.84% had COPD, 22.47% had bronchiectasis, 31.46% were diabetic, 15.73% were on long term steroid use and 14.61% had history of prior hospital admission.

Conclusion: Age, smoking, structural lung diseases like COPD, bronchiectasis, long term steroid use and prior hospital admission are important risk factors for *Pseudomonas* pneumonia.

Keywords: Community acquired pneumonia, Diabetes in pneumonia, Multidrug resistant pneumonia, Risk factors

INTRODUCTION

Community Acquired Pneumonia (CAP) is an important cause of the morbidity and mortality all over the world [1]. It is estimated that around 5-6 billion people are diagnosed with CAP and more than 3.5 million people succumb to death yearly due to CAP [1,2]. *P. aeruginosa* is a gram negative, aerobic rod bacterium which belongs to the Gamma Proteobacteria bacterial class [3]. This microbe is commonly seen on the skin of humans and hence considered as skin flora. It is an opportunistic pathogen which respond to a breakdown of the skin and enter the body of immune-compromised individuals [4]. Soil, water, agriculture plants, animals, and humans are usual reservoirs of this microbe [5].

P. aeruginosa has two modes of virulence expression, resulting in atleast two forms of distinct pathogenetic behaviour:

- Some strains remain confined to the lungs as a chronic, indolent coloniser {as occurs in many patients with Cystic Fibrosis (CF)}.
- Other strains can invade tissues, causing pneumonia or bacteremia along with their potential complications of septic shock and death.

P. aeruginosa is one of the common causative organisms for both hospital-acquired pneumonia [6] and CAP. Although community acquired *P. aeruginosa* pneumonia is occasionally reported in otherwise healthy hosts [7], most patients have an identifiable risk factor for disease. The various risk factors that have been identified include structural lung diseases such as CF, bronchiectasis, compromised immune system (as in Human Immunodeficiency Virus (HIV) infected patients, those on immunosuppressive agents, neutropenic hosts), repeated exacerbation of COPD and recurrent use of glucocorticoids and antibiotics. Other known risk factors for *Pseudomonas* infection include cirrhosis, enteral tube feeding, intubation and prior hospital admission. Risk factors associated with hospital-acquired *Pseudomonas* infection include increasing age, duration of mechanical ventilation, previous use of antibiotics, admission to a high dependency unit or Intensive Care Unit (ICU), and admission in a ward with higher incidence of patients with *P. aeruginosa* infections [8]. Outbreaks of *P. aeruginosa* infection linked to contaminated healthcare equipment such as endoscopes have been occasionally reported [9].

Signs and symptoms of *P. aeruginosa* pneumonia are similar to that caused by other pyogenic bacteria or *Legionella*, and there are no features specific to *P. aeruginosa* which can reliably distinguish it from pneumonia caused by other organisms. Characteristic symptoms of acute *P. aeruginosa* pneumonia are cough productive of purulent sputum, fever, chills, dyspnoea, confusion and severe systemic toxicity. Ventilator-associated *P. aeruginosa* pneumonia patients may also present with increased tracheobronchial secretions and decreased ventilator performance, which can develop suddenly or gradually.

Radiographic findings associated with *P. aeruginosa* pneumonia are variable and there is no unique finding which is predictive or characteristic of the disease. It may present as diffuse bilateral infiltrates, with or without pleural effusion. Many patients may present with multifocal airspace consolidation. Other radiographic features include nodular infiltrates, tree-in-bud opacities, and necrosis [10]. Sometimes, areas of radiolucency suggestive of cavitary disease can be seen. However, classic lobar consolidation is uncommon. In case of *P. aeruginosa* pneumonia arising from haematogenous spread of the organism, the early radiographic findings may include pulmonary congestion and interstitial oedema. Later these patients

often develop diffuse interstitial and alveolar infiltrates after 24 to 48 hours. Large haemorrhagic nodules with central necrosis or cavities are very rare in the course of the disease [11].

Histopathological changes seen in patients with *P. aeruginosa* pneumonia usually include microabscesses with focal haemorrhage and necrosis of the alveolar septae without evidence of bacterial invasion of vessel walls or vascular necrosis. In patients with pneumonia due to haematogenous spread, the pathologic changes usually include intraalveolar haemorrhage and necrosis around pulmonary vessels. In some patients, small (2-15 mm), yellow-brown, firm, necrotic nodules with areas of dark red haemorrhagic parenchyma may also be seen along with liquefactive necrosis or bacterial invasion of the alveolar cell walls [12].

Specifically, for the empiric management of CAP and hospital-acquired pneumonia, the guidelines from the Infectious Diseases Society of America and the American Thoracic Society are followed. The following antimicrobial combinations are recommended for patients with risk factors for both *P. aeruginosa* infection as well as drug resistance [13]:

- An antipseudomonal beta-lactam PLUS an antipseudomonal quinolone
- An antipseudomonal beta-lactam PLUS an aminoglycoside
- An antipseudomonal quinolone PLUS an aminoglycoside

P. aeruginosa pneumonia can lead to high in-hospital mortality rates and prolonged duration of hospital stay [14, 15]. Now-a-days, circulating strains of *P. aeruginosa* with higher resistance patterns to antipseudomonal antibiotics have emerged, resulting in infections that are very challenging to treat [6].

The knowledge regarding specific risk factors associated with *Pseudomonas* pneumonia helps in the early diagnosis and prompt initiation of empirical treatment. This study was an attempt to throw more insight regarding various risk factors associated with *P. aeruginosa* infection. Also, this study was done to assess certain factors which are less studied as potential risk factors for *Pseudomonas* infection.

MATERIALS AND METHODS

This was an observational, cross-sectional, hospital based study conducted in Jawaharlal Nehru Medical College, Aligarh, which is a tertiary care centre in the northern state, Uttar Pradesh, India. The participants were from the Department of Respiratory Medicine of the same hospital and the study data was collected from November 2018 to November 2020. The Institutional Ethics Committee approved the study (letter no: 260/FM/AMU).

Inclusion criteria: All patients with diagnosis of *Pseudomonas* pneumonia, defined as the presence of signs and symptoms of pneumonia with pulmonary infiltrates suggestive of pneumonia on thoracic imaging along with microbiological confirmation (sputum/tracheal culture positive) for *P. aeruginosa* were considered for the study. Among these patients those who gave informed consent and were above 18 years of age were included in the study.

Exclusion criteria: Patients suffering from other respiratory infections such as pulmonary tuberculosis, Coronavirus Disease (COVID)/viral pneumonia and other bacterial infections were excluded from the study.

Methodology

A total of 89 patients with *P. aeruginosa* pneumonia were enrolled in the study and they were assessed with thorough history and clinical examination. A pretested interviewer administered questionnaire was used to obtain relevant information from the study population. Any significant risk factor for *P. aeruginosa* was assessed from the history like smoking, alcohol intake, diabetes mellitus, bronchiectasis, COPD, previous hospital admissions, recurrent glucocorticoid use, recent bronchoscopy/intercostal tube drainage, tracheostomy and congenital heart diseases. All these patients were thoroughly examined for any respiratory/cardiovascular/gastrointestinal/central nervous system abnormalities. Any systemic abnormalities noted other than respiratory abnormalities were referred to speciality departments for expert opinion.

All recruited participants were subjected to routine blood investigations, spirometry and chest X-ray. The following investigations were done for all participants like haemogram, renal fuction test, liver function test, blood glucose -fasting and postprandial, glycated Haemoglobin (HbA1c), Electrocardiogram (ECG), chest X-ray, Pulmonary Function Tests (PFT)- pre and post bronchodilator and HIV Enzyme Linked Immunosorbent Assay (ELISA). Additional investigation like High Resolution Computed Tomography (HRCT)/Contrast Enhanced Computed Tomography (CECT), were done when chest X-ray showed any abnormality.

STATISTICAL ANALYSIS

Data analysis was done using SPSS version 22.0. Descriptive statistics was done and the qualitative data were expressed in percentage. Categorical variables were compared with the Chi-square test. All tests were performed at 5% level of significance. All reported p-values were two tailed, p-value of <0.05 was considered significant, while p-value of <0.01 was considered highly significant.

RESULTS

A total of 89 patients with *Pseudomonas* pneumonia were enrolled in the study, out of which 55 were males and 34 were females.

Age: Most of the patients were above 40 years. The data was statistically significant and showed that the incidence of *Pseudomonas* pneumonia increased with age. A 77.5% of the patients were above 40 years of age [Table/Fig-1].

Smoking: This study revealed that 33 (37.08%) patients out of the total 89 patients were significant cigarette/beedi smokers. Significant smoking was taken as smoking index more than 100 (smoking index=no of cigarettes per day×years of smoking) [Table/Fig-2].

Age (years)	No. of cases	Percentage	p-value*
18-40	20	22.5	<0.05
41-60	37	41.6	
>60	32	35.9	
Total	89	100	

[Table/Fig-1]: Shows age distribution.
*Chi-square test; p-value <0.05 considered significant

COPD: Among the patients with *Pseudomonas* pneumonia, 23 patients were diagnosed with or previously diagnosed with COPD. All subjects underwent PFT and Chest X-ray at the time of admission. COPD was considered in those subjects in whom Forced Expiratory Volume during one second (FEV1)/Forced Vital Capacity (FVC) <0.7 and relevant clinical history. Chest X-ray findings like hyperinflated lung fields, flattened diaphragm, tubular heart were considered as auxiliary findings to support the diagnosis of COPD [Table/Fig-2].

Risk factors	Number of cases (%)		p-value*
	Yes	No	
Smoking	33 (37.08)	56 (62.92)	<0.05
COPD	23 (25.84)	66 (74.16)	<0.001
Bronchiectasis	20 (22.47)	69 (77.53)	<0.001
Chronic steriod use	14 (15.73)	75 (84.27)	<0.001
Prior hospital stay	13 (14.61)	76 (85.39)	<0.001
Diabetes mellitus	28 (31.46)	61 (68.54)	<0.001

[Table/Fig-2]: Percentage of *P. aeruginosa* pneumonia patients with/without the risk factors.
*Chi-square test; p-value <0.05 considered significant

Bronchiectasis: Total 20 (22.47%) patients were detected to have bronchiectasis, of the total 89 study participants. Bronchiectasis was

considered in those patients with relevant history and radiological features. Those patients with suspicious chest X-ray findings of bronchiectasis were confirmed with HRCT [Table/Fig-2].

Chronic steroid use: Total 14 patients (15.73%) were on long term steroid for various medical conditions like arthritis, bronchial asthma etc. Chronic steroid use was considered as any use of steroid drugs for a period of more than two months. It included the use of inhaled corticosteroids used for the treatment of respiratory diseases [Table/Fig-2].

Prior hospital stay: Total 13 patients (15.61%) of the study population had prior hospital admission atleast for one day within 90 days of diagnosis. Prior hospital admissions for these subjects were done for both respiratory and non respiratory conditions [Table/Fig-2].

Diabetes mellitus: Total 28 patients (31.46%) were newly/previously diagnosed cases of type 2 diabetes mellitus. HbA1c value more than 7.5 was taken as the cut-off for the subjects. In those patients with HbA1c between 7-7.5, any blood glucose value more than 180 mg/dL were considered diabetic [Table/Fig-2].

DISCUSSION

Pseudomonas aeruginosa is a gram negative bacterium which can cause life-threatening hospital acquired respiratory infections [2]. Now-a-days, CAP caused by *Pseudomonas* is also on the increasing trend. Although the true prevalence of *P. aeruginosa* CAP is unknown, a meta-analysis done by Chalmers JD et al., reported data from 22 studies showed a prevalence ranging from 0-23% in different CAP populations [16]. *P. aeruginosa* pneumonia often results in severe illness and poor clinical response to treatment [6]. This study aimed to determine various risk factors associated with *Pseudomonas* pneumonia and it was found that increasing age, smoking, diabetes mellitus, structural lung diseases like COPD, bronchiectasis, chronic use of steroids and previous hospital admission within in a period of 90 days were some of the risk factors associated with *P. aeruginosa* pneumonia.

Out of 89 patients with *Pseudomonas* pneumonia, 77.5% were above 40 years of age, 37.08% were smokers, 25.84% had COPD, 22.47% had bronchiectasis, 31.46% were diabetic,15.73% were on long term steroid use and 14.61% had history of prior hospital admission. Increasing age was found to be a predisposing factor for *P. aeruginosa* pneumonia in the study and it must be due to weakening of immunity as the age advances. Cigarette smoking impairs mucociliary clearance at the same time causing an increase in mucus production which might be the reason for it being a risk factor. COPD causes destruction of portions of airway, increased mucus production and chronic inflammation which increases the risk for *P. aeruginosa* pneumonia. Similarly structural damage caused by bronchiectasis leads to stasis of secretions, which might have increased the risk. Diabetes mellitus and long tern steroid use leads to immune suppression, probably the reason for their association with *P. aeruginosa* pneumonia in the study. Previous hospital admissions expose the individuals to nosocomial microbes and hence predispose to *Pseudomonas* pneumonia.

The antibiotic treatment for *P. aeruginosa* differs from the standard treatment protocol for pneumonia that targets the most common microorganism causing CAP (e.g., *Streptococcus pneumoniae*), and is extrapolated from Ventilator-Associated Pneumonia (VAP) and Health Care-Associated Pneumonia (HCAP) data. As per the recent guidelines for CAP in adult patients, *P. aeruginosa* empirical antibiotic treatment is recommended only in patients with specific clinical risk factors [16]. These risk factors for CAP due to *P. aeruginosa* in current clinical practice guidelines include smoking, COPD, bronchiectasis, long term use of oral steroids or any prior use of antibiotics within 90 days [17].

Sibila O et al., did a retrospective population based study in which 781 patients with *P. aeruginosa* pneumonia were identified in a cohort of 62,689 patients (1.1%) [18]. They summarised that risk factors recommended by current guidelines were only able to detect one third of the patients with CAP due to *P. aeruginosa*. They suggested that increasing age is a risk factor for *Pseudomonas* pneumonia, probably because of weakened immunity.

Chien J et al., also supported present study finding and suggested that cigarette smoke exposure not only increases the virulence of *P. aeruginosa* but can also impair the neutrophil mediated killing efficacy [19]. Their study also opined that exposure to cigarette smoke increases *Pseudomonas* biofilm formation which may be a reason for increased risk of *Pseudomonas* infection. Hatchette TF et al., suggested that pack years of more than 40 is a significant risk factor for *Pseudomonas* pneumonia [20].

The study conducted by Lieberman D and Lieberman D, among unselected outpatients with acute exacerbations of COPD revealed that average rate of isolation of *P. aeruginosa* from the patients' sputum was around 4%. This rate was even higher in COPD patients with severe airflow obstruction, in whom the rate of isolation of *P. aeruginosa* in sputum reached 8-13% of all episodes of acute exacerbations of COPD [21]. The study conducted by Garcia-Vidal C et al., showed that out of 188 COPD patients included, 31 (16.5%) had *P. aeruginosa* in sputum at initial admission [22]. The most important finding of their study was the strong correlation between *Pseudomonas* isolation at hospital admission and various markers of respiratory functional impairment used in COPD. The various markers of respiratory functional used were BODE index (BMI, Airflow Obstruction, Dyspnoea, Exercise Capacity), modified Medical Research Council (mMRC) grading of Dyspnoea, Home Oxygen therapy requirement etc. Their study concluded that acute exacerbation of COPD must be considered as high risk condition for *Pseudomonas* infection and antibiotics should be started accordingly [22].

Restrepo MI et al., confirmed present study findings suggesting bronchiectasis (OR 2.88, 95% CI 1.65-5.05) as one of the main risk factor for *Pseudomonas* infection. Their study also concluded that patients with bronchiectasis had increased chance of getting infection with multidrug resistant *P. aeruginosa* [23]. In an observational study, conducted by Pieters A et al., among patients with bronchiectasis, they identified patient characteristics which were associated with the presence of persistent *P. aeruginosa* colonisation [24]. They stated that *P. aeruginosa* is an important pathogen in bronchiectasis and functions as a marker for disease severity. As per their study, chronic colonisation with *P. aeruginosa* were seen in 25% of the patients with bronchiectasis.

As per the study conducted by Garcia-Vidal C et al., 31 patients were culture positive for *Pseudomonas* infection, out of which 4 patients (12.9%) were on chronic steroid use [22]. Sibila O et al., documented that 27.1% of *Pseudomonas* infection patients were on chronic steroid use [18]. Raman G et al., did a study which concluded that prior antibiotics use and previous hospital or ICU admissions were the most important risk factors for the development of resistant *P. aeruginosa* infections. They also stated that the risk of development of multidrug resistant *P. aeruginosa* compared with non-*P. aeruginosa* was significantly increased with prior use of cephalosporins (OR 3.96), carbapenems (OR 2.61), quinolones (OR 2.96), and prior hospital stay (OR 1.74) [25].

Sibila O et al., also stated that diabetes mellitus can be a risk factor for *Pseudomans* pneumonia [18]. Saibal MAA et al., suggested that a wide range of neutrophil and macrophage functions are impaired in diabetes mellitus [26]. These include chemotaxis, adherence, phagocytosis and the ability to kill the phagocytosed microorganism. The intracellular killing of microbes by free radicals, called respiratory burst are also reduced in diabetes. Gradual impairment of acquired immunity can also occur in diabetic patients. As a result of chronic hyperglycaemia, there will be alterations in the capillary endothelial function, Red Blood Cells (RBCs) rigidity and changes in the oxygen dissociation curve which may affect the hosts' ability to combat infections.

Limitation(s)

This was an observational, one point study done only in those patients who were having *P. aeruginosa* pneumonia. Since it is not a case-control study; the odd's ratio was not calculated. Many confounding factors might have affected the results. For example, in the study a patient who is a heavy smoker might also be having borderline diabetes mellitus. This could result in smoking being a confounding factor for diabetes or vice-versa. The confounding factors were not filtered.

CONCLUSION(S)

This study reveals important information regarding predisposing factors of *Pseudomonas* pneumonia. Seven risk factors were identified including increasing age, smoking, chronic obstructive lung disease, bronchiectasis, chronic steroid use, diabetes mellitus and prior hospital stay. The knowledge about these predisposing factors helps in suspecting *P. aeruginosa* infection at the earliest and prompt initiation of empirical antipseudomonal antibiotics.

REFERENCES

[1] Prina E, Ranzani OT, Torres A. Community-acquired pneumonia. Lancet. 2015;386:1097-108.
[2] Wunderink RG, Waterer GW. Community-acquired pneumonia. N Engl J Med. 2014;370:1863.
[3] Marrie TJ, Beecroft MD, Herman-Gyidic Z. Resolution of symptoms in patients with community acquired pneumonia treated on an ambulatory basis. J Infect. 2004;49:302-09.
[4] Todar K. Online Textbook of Microbiology. Oppurtunistic infections caused by Pseudomonas aeruginosa. Retrieved March 14, 2010, from http://www.textbookofbacteriology.net/pseudomonas.html.
[5] Qarah S, Cunha BA, Dua P, Lessnau K. (2009, December 9). Pseudomonas aeruginosa infections. Retrieved March 13, 2010, from http://emedicine.medscape.com/article/226748-overview.
[6] Weber DJ, Rutala WA, Sickbert-Bennett EE, Samsa GP, Brown V, Niederman MS. Microbiology of ventilator-associated pneumonia compared with that of hospital-acquired pneumonia. Infect Control Hosp Epidemiol. 2007;28:825.
[7] Henderson A, Kelly W, Wright M. Fulminant primary *Pseudomonas aeruginosa* pneumonia and septicaemia in previously well adults. Intensive Care Med. 1992;18:430.
[8] Venier AG, Gruson D, Lavigne T, Jarno P, L'hériteau F, Coignard B, et al. Identifying new risk factors for *Pseudomonas aeruginosa* pneumonia in intensive care units: Experience of the French national surveillance, REA-RAISIN. J Hosp Infect. 2011;79:44.
[9] Kirschke DL, Jones TF, Craig AS, Chu PS, Mayernick GG, Jayesh RN, et al. *Pseudomonas aeruginosa* and *Serratia marcescens* contamination associated with a manufacturing defect in bronchoscopes. N Engl J Med. 2003;348:214.
[10] Shah RM, Wechsler R, Salazar AM, Spirn PW. Spectrum of CT findings in nosocomial *Pseudomonas aeruginosa* pneumonia. J Thorac Imaging. 2002;17:53.
[11] Schuster MG, Norris AH. Community-acquired *Pseudomonas aeruginosa* pneumonia in patients with HIV infection. AIDS. 1994;8:1437.
[12] Bonifacio SL, Kitterman JA, Ursell PC. *Pseudomonas pneumonia* in infants: An autopsy study. Hum Pathol. 2003;34:929.
[13] American Thoracic Society, Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171:388.
[14] Crabtree TD, Gleason TG, Pruett TL, Sawyer RG. Trends in nosocomial pneumonia in surgical patients as we approach the 21st century: A prospective analysis. Am Surg. 1999;65:706.
[15] Fujitani S, Sun HY, Yu VL, Weingarten JA. Pneumonia due to *Pseudomonas aeruginosa*: Part I: Epidemiology, clinical diagnosis, and source. Chest. 2011;139:909.
[16] Chalmers JD, Rother C, Salih W, Ewig S. Healthcare-associated pneumonia does not accurately identify potentially resistant pathogens: A systematic review and meta-analysis. Clin Infect Dis. 2014;58:330-39.
[17] Mandell LA, Wunderink RG, Anzueto A, Bartlett JG, Campbell GD, Dean NC, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines for the management of community acquired pneumonia in adults. Clin Infect Dis. 2007;44:S27-72.
[18] Sibila O, Laserna E, Maselli DJ, Fernandez JF, Mortensen EM, Anzueto A, et al. Risk factors and antibiotic therapy in P.aeruginosa community-acquired pneumonia. Respirology. 2015;20:660-66. https://doi.org/10.1111/resp.12506.
[19] Chien J, Hwang JH, Nilaad S, Masso Silva JA, Ahn SJ, McEachern EK, et al. Cigarette smoke exposure promotes virulence of Pseudomonas aeruginosa and induces resistance to neutrophil killing. Infect Immun. 2020;88(11):e00527-20. Doi: 10.1128/IAI.00527-20.
[20] Hatchette TF, Gupta R, Marrie TJ. *Pseudomonas aeruginosa* community-acquired pneumonia in previously healthy adults: Case report and review of the literature. Clinical Infectious Diseases. 2020;31(6):1349-56. https://doi.org/10.1086/317486.
[21] Lieberman D, Lieberman D. Pseudomonal infections in patients with COPD: Epidemiology and management. Am J Respir Med. 2003;2(6):459-68.
[22] Garcia-Vidal C, Almagro P, Romaní V, Rodríguez-Carballeira M, Cuchi E, Canales L, et al. Pseudomonas aeruginosa in patients hospitalised for COPD exacerbation: A prospective study. Eur Respir J. 2009 Nov;34(5):1072-8. Doi: 10.1183/09031936.00003309. Epub 2009 Apr 22. PMID: 19386694.
[23] Restrepo MI, Babu BL, Reyes LF, Chalmers JD, Soni NJ, Sibila O, et al. Burden and risk factors for *Pseudomonas aeruginosa* community-acquired pneumonia: A multinational point prevalence study of hospitalised patients. Eur Respir J. 2018;52:1701190.
[24] Pieters A, Bakker M, Hoek RAS, Altenburg J, van Westreenen M, Aerts JGJV, et al. Predicting factors for chronic colonisation of *Pseudomonas aeruginosa* in bronchiectasis. Eur J Clin Microbiol Infect Dis. 2019;38:2299-304.
[25] Raman G, Avendano E, Chan J, Merchant S, Puzniak L. Risk factors for hospitalized patients with resistant or multidrug-resistant *Pseudomonas aeruginosa* infections: A systematic review and meta-analysis. Antimicrob Resist Infect Control. 2018;7:79.
[26] Saibal MAA, Rahman SHZ, Nishat L, Sikder NH, Begum SA, Islam MJ, Uddin KN. Community acquired pneumonia in diabetic and non-diabetic hospitalized patients: Presentation, causative pathogens and outcome. Bangladesh Medical Research Council Bulletin. 2012;38(3):98-103.

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