ISSN 2664-4169 (Print) & ISSN 2664-7923 (Online)
South Asian Research Journal of Biology and Applied Biosciences
Abbreviated Key Title: South Asian Res J Bio Appl Biosci

| Volume-7 | Issue-6 | Nov-Dec -2025 | **DOI: https://doi.org/10.36346/sarjbab.2025.v07i06.001**

Review Article

Epidemiology, Virulence Factors and Treatment for Klebsiella Pneumoniae: Review Article

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Article History
Received: 04.09.2025
Accepted: 27.10.2025
Published: 01.11.2025

Abstract: Klebsiella pneumoniae is community-acquired infection it can present as lung tuberculosis. It is difficult to treat K. pneumoniae because of its enlarged capsule. "The most effective treatments for Klebsiella are 3&4 century carbapenems, quinolones, or cephalosporins." With the use of more recent medications, single therapy is similarly beneficial as combination therapy for lung infection. Successful treatment in the past has involved the use of older medicines that had less Klebsiella involvement. Prior to identification of pneumococcus illness, patient effect to tuberculosis, and ceftriaxone monotherapy was the prescribed treatment. The patient received an injection at first, followed by three weeks of oral medication. The patient received an injection at first, followed by 3 weeks of oral medication. This article's goal is to discuss the epidemiology, virulence factors, and treatment approaches of this particular species of bacterium. The bacteriais widely distributed throughout the nation, causing a number of respiratory illnesses and having the ability to transmit to other pathogens, including viruses, especially the Corona virus.

Keywords: Taxonomy, Klebsiella Pneumonia, Epidemiology, Virulence, Polysaccharides.

1. INTRODUCTION

The K. pneumoniae Non-motile, encapsulation-fermenting, gram-negative bacteria that can optionally be anaerobic. It causes bacterial pneumonias that are quite small. It could result in significant lung consolidation caused by hemorrhage and necrotization. it causes focal bacteremia and urinary tract infections in individuals who are already impaired [1]. Hospital infections frequently have a connection to this M.o. Conditions like cancer, Immunity can be weakened by liver failure, bile disorders, infections of the bladder, insulin resistance, bone cancer, bacteremia, and alcoholism and increase risk catching K. pneumoniae. The most common cause of GNB is Escherichia coli, which is followed by this species. "In general, K. pneumoniae bacteremia is the cause of illness and death. The main characteristics of K. pneumoniae are spreading infections, such as brain abscess, eye infections and encephalitis. " [2]. Although it demonstrated that (m.o) can formation a biofilm in vitro since the late 1981s, Reid and his associates did not examine the bladder cells patient who had a spinal cord K. pneumoniae infection until 1992 [3]. About 41% of K. pneumoniae were able to produce biofilms from mucus, fluids such as blood and urine, and swab from trauma, according to later in vitro studies [4].

2. Genus Klebsiella

Klebsiella is a one of the Enterobacteriaceae. It has name of Edwin Klebs, a German microbiologist who lived from 1834 to 1913 [6]. Klebsiella is a ubiquitous organism found in environment. This is because several lines of descent carve out different spaces variants that are more climate-appropriate due to corresponding biochemical modifications. Water, soil, plants, insects, animals, and people all contain it [6]. They are usually straight rods with ends that are round or pointed. It shown alone pairs or short chains [7], and depending on the structure and pressure of the medium [8], it can generate colonies that are glossy and somewhat dome-shaped, with varied degrees of stubbornness. Klebesiella spp are

typically normal flora in the human nose, throat, and gastrointestinal system, they can also behave as opportunistic human pathogens [8].

3. Klebsiella Taxonomy

States that biochemical reactions are typically used to identify and differentiate Klebsiella species. in line with the Enterobacteriaceae family's overall description. Non-motile (except for K. mobilis), gram-negative, potentially anaerobic, with a fermentation, respiratory like, & negative oxidase A popular encapsulated bacilli that typically tests positive in the Voges-Proskauer test and produces lysine decarboxylase but not decarboxylase ornithine.

4. Klebsiella Pneumoniae

Friedlander isolated K. pneumoniae from pneumonia patients' lungs for the first time in 1882. Originally called the bacillus of Friedlander, this encapsulated bacterium changed its name Klebsiella in 1886. Afterwards, it was discovered to be a saprophyte bacteria that infects more than simply the human outermost layer, nasal passages, and gut [2]. About 5% of healthy people have K. pneumoniae in their stools and respiratory system. It is responsible for about 1 percent of bacterial pneumonia cases. One method by which K. pneumoniae Hemorrhagic necrotic damage is one way to accomplish substantial lung aggregation. It can occasionally result in UTI & infection in blood with localized lesions in patients who are already impaired. Pneumonia can also result from certain enterics. Hospital infections are caused by K. pneumonia and K. oxytoca [1]. K. pneummiae, a verdure of the human digestive system, is occasionally linked to contamination acquired in a clinic. Risk, liver disease, bile sac lot problems, osteomyelitis, bacteremia, diabetes mellitus, and alcohol abuse are among hidden illnesses that might impair a person's defenses and raise the possibility of contracting K. pneumoniae. After Escherichia coli, K. pneumoniae is the 2nd more important cause of (G -ve) bacteremia. In all populations, K. pneumoniae bacteremia results in notable mortality and depressing conditions. The primary features of K. pneumoniae infections are their ability to appear manifestations, such as endophthalmitis, meningitis, and pyogenic cerebrum boil [9].

5. Epidemiology

Humans are K. pneumoniae's primary host. One to six percent of the general population has the organism in their nasopharynx, and 5 to 38 percent have it in feces. The Gut tract & hospital main source of infection is worker hands. Nosocomial eruptions may result from it. However, higher colonization rates have been observed by people of Chinese heritage & suffer from persistent drinking. The prevalence of K. pneumoniae carriers is higher in hospitalized patients than in the general population. It is possible to observe and feel that hospitalized patients' stool contains up to 75% carriers, which to dosages of antibiotics administered [10, 11].

6. Theme of Virulence

Researchers have looked for bacterial factors that are similar to the pathogenesis of Klebsiella infections [12].

1. Capsules

The mucoid is produced by the normally thick hydrophilic polysaccharide capsule that envelops the Klebsiella strains., sparkling appearance of agar colonies [6]. Numerous host defensive mechanisms cannot penetrate this capsule [12]. In subcultures, the harm caused by this trait was associated with low in virulence [6]. Found capsule has been found to greatly reduce bacterial phagocytosis with macrophages and impede the elements of the bacterial complement are dropped in vitro. [13]. Additionally, the production of capsules may result in transcriptional suppression in another adhesive and hinder the suitable Type 1 fimbria formation on the outermost layer of the bacteria [14]. capsule-negative fragments had a higher adhesion to cells grown in conjunction with wild-type strains [15].

2. Lipopolysaccharides

O-antigen is retain K. As capsular or non-capsular strains that do not have the O1 antigen, pneumoniae from accompanied fatalities are particularly vulnerable to the bactericidal effects of conventional and additional supplemental routes [16]. But in non-immune serums, opsonizing allows K-O+ phagocytosis-prone bacteria, and O-antigen is exceptionally good at turning on the initial elements. Reports of protective antibodies against the extracellular toxic complex's (ETC) lipopolysaccharide (toxicity) component have surfaced [17].

3. Adhesins

Attachment to the surfaces of mucosa and epithelial cells is frequently initial stage of colonization and infection. Bacterial cell surface fimbriae may include adhesins, which are also hemagglutinins. Spp of K pneumonia & K oxytoca can form type-1, massive, channeled fimbriae that resemble those of other Enterobacteriaceae.The cause of D-mannose-sensitive hemagglutination is Klebsiella type 1 [18]. Additionally, ciliated in vitro tracheal cells interact with this fimbria [19].

4. Synthesis of Biofilm by K. Pneumoniae

Microorganisms adhere cells to a surface is called a biofilm. These adherent Cells are often immersed in a coating of polymers that are extracellular (EPS) that they have created on their own. The biofilm although goo is not a biofilm,

extracellular polymers, often known as slime, are a polymeric buildup that is usually made up of extracellular proteins, polysaccharides, and DNA [21]. Certain species are unable to adhere to their own surface, yet they can frequently adhere either directly to previous colonists or to the matrix. Cells use quorum sensing to interact with substances like acylated homoserine lactone (AHL) during this invasion. Some bacteria are less able to produce biofilms due to their limited motility. Compared to motile bacteria, nonmotile bacteria are less able to differentiate or accumulate on the surface [22]. The process of biofilm formation involves the following key stages:

A. Reversible Connection

Numerous physical, chemical, and biological events took place on the bacterial surface as a result of this initial encounter. Van der Waals, hydrophobic forces, and electrostatic forces are examples of non-specific interactions that usually aid in the basic bacteria-surface attaching on the abiotic surface. In contrast, lectin or sticky, two examples of complex molecular docking processes, are used to accomplish biotic surface binding, such as tissue [23]. According to other research, planktonic cells may move to make their first surface contact with bacteria and abiotic surfaces [24].

B. Irreversible Attachment

Bacterial cells begin their period of irreversible attachment, growth, and aggregation as multi-layered cell classes after the exopolymer-led surface authority. These extracellular grids—which consist of a variety of resources like polypeptide, NA, complex carbohydrates, & other components—are thought to be essential for keeping microbial cells together in the polymeric structure, assisting in the capture & retention of nutrients in biophilm formation, shielding cells from desiccation, and the impact of antibiotic experts [25].

C. Biofilm Development

Following their irreversible commitment to a surface, the bacterial cells change phenotypically, & the biofilm development starts. Either by clonally developing, recruiting planktonic or bulk fluid cells, or aggregating already secured cells, bacteria start to create micro-colonies. The connected cells produce a variety of extracellular elements that combine with external chemical and inorganic substances to generate glycocalyx [26-27]. It was suggested that, similar to how tissues comprise more complex organisms, the microcolony serves as the primary unit of biofilm growth. Similar to those in higher species, the biofilm's liquid pathways function as an embryonic circulatory pathway. The spatial and temporal structuring of microbial biofilms is protective. Since bacteria receive nutrients through water channels at a low water flow rate [28]. In vitro biofilm formation of K. pneumoniae has been documented since the late 1980s.

7. Treatment

Since K. pneumonia is not common When treating pneumonia in people of all ages, normal antibacterial regimens should be Follow after infection Following suspicion or evidence of a K. pneumoniae infection, antimicrobial medication should be tailored to local antimicrobial therapy protocols. [33]. A 14-day course of medication consisting of a 3 - or 4 - group of cephalosporin, respiratory quinolone, or an aminoglycoside from one or both of the aforementioned regimens is the accepted treatment for acquired community pneumonia at the moment. Azotreonam or quinolone in the air should be administered if the patient is allergic to penicillins. Prior to the identification of sensitivities, for infections that occur in the hospital, carbapenem therapy can be used as a single [34–35].

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