

KLEBSIELLA PNEUMONIA *by Bill Wojciechowski, MS, RRT*



Klebsiella pneumonia, or Friedländer's pneumonia, is caused by the microorganism *Klebsiella pneumoniae*. The genus *Klebsiella* belongs to the tribe *Klebsiellae*, which is a member of the family *Enterobacteriaceae*. The *Klebsiella* microorganisms were named after the German microbiologist named Edwin Klebs who lived in the 19th century, and studied this genus.

Bacteria have characteristic shapes (cocci, rods, spirals, etc.), and often occur in aggregates (pairs, chains, tetrads, clusters, etc.). These traits are usually typical for a genus and are diagnostically useful. *Klebsiellae* are non-motile, rod-shaped, gram-negative, facultative anaerobic bacteria. Facultative bacteria produce ATP via aerobic respiration when oxygen is available, but are capable of switching to fermentation to create ATP anaerobically.

Bacterial Cell Wall – Biochemical Structure

The biochemical structure of the bacterial cell wall is uniform. It consists of a single continuous molecule, the peptidoglycan. A complete peptidoglycan molecule is comprised of numerous sugar molecules cross-linked to each other, creating a fence-like structure that encloses the fragile protoplast. The protoplast (spheroplast) is the living material of a bacterial cell, including the protoplasm and plasma membrane, excluding the cell wall.

Gram-negative bacteria have a more complex cell wall than gram-positive bacteria. Gram-negative bacteria appear to have a bi-layered cell wall. The inner layer of the cell wall is the pepti-

doglycan molecule with its cross-linked sugar molecules. The outer component of the cell wall is often referred to as the outer membrane because of its structural and chemical resemblance to biological membranes. The outer membrane of a gram-negative bacterium is mainly composed of lipopolysaccharide (LPS) molecules. These LPS molecules have toxic activity, and are responsible for many symptoms associated with infections caused by gram-negative species, e.g., vascular collapse, hemorrhage, shock, and leukopenia. Because LPS molecules are the main determinant of the pathogenicity and the virulence in gram-negative species, LPSs are often referred to as endotoxins. The lipopolysaccharide layer, due to its thick, gelatinous nature, also confers resistance to phagocytosis by polymorphonuclear leukocytes, and protects *K. pneumoniae* from cellular death caused by bactericidal serum factors. Located along the outer component of the bi-layer of *K. pneumoniae* are pili, hair-like appendages that assist the microorganism to adhere to cells in the host, and enhance the organism's ability to colonize the host.

Epidemiology

K. pneumoniae is a normal resident of the intestinal flora in 40% of the population. Oropharyngeal colonization can develop from endotracheal intubation. *Klebsiella pneumoniae* rarely afflicts healthy hosts. *K. pneumoniae* is a ubiquitous microorganism that tends to affect middle-aged and elderly males who have chronic conditions such as alcoholism, diabetes mellitus, and COPD. Immunocompromised hosts are also at increased risk.

As a class of microbes, gram-negative bacilli cause 3% to 10% of community-acquired pneumonias. *Klebsiella* is responsible for less than 1%. Despite this extremely low occurrence, *Klebsiella pneumoniae* is associated with a 50% mortality rate. In patients who develop *Klebsiella pneumoniae* and bacteremia because of alcoholism, the mortality rate jumps to almost 100%.

The *Klebsiella* genus is notorious for causing many hospital-acquired infections such as pneumonia, bacteremia, thrombophlebitis, urinary tract infection, upper respiratory tract infection, and diarrhea. *Klebsiella* infections are far more prevalent in health care settings than in the community. *K. pneumoniae* is responsible for most urinary, gastrointestinal, and respiratory tract infections and account for approximately 8% of all nosocomial infections. Being ubiquitous, *K. pneumoniae* is found throughout the environment. It has been cultured from soil and water.

Pathophysiology

Klebsiella enters the host when the host aspirates colonizing microorganisms from the oropharyngeal region into the lower respiratory tract. It has a predilection for the upper lobes of the lungs. However, infection of the lower lobes is not uncommon.

In addition to causing intrapulmonary shunting, *Klebsiella pneumoniae* is a necrotizing disease process, as evidenced by alveolar wall destruction. As a sequela to the *Klebsiella* infection, puss associated with the abscesses can produce an empyema as well. Timely evacuation of an empyema is critical to avoid trapping of pus in the pleural space, development of adhesions between the parietal and visceral pleurae, and formation of scar

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tissue. Inflammation and hemorrhage occur within the lung tissue, with thick, bloody sputum.

Signs and Symptoms

Klebsiella pneumonia characteristically occurs in males, who have a history of alcoholism and are over 48 years of age. Chest radiography frequently demonstrates cavity formation and swelling of the affected lobe, which causes bulging of the interlobar fissures.

The classical clinical presentation of Klebsiella pneumonia includes sudden onset, prostration, high fever, chills, flu-like symptoms, and hemoptysis. Pleuritic chest pain and dyspnea are also involved. The sputum produced by infected patients is thick, bloody, and mucoid, and is commonly described as resembling currant jelly.

Physical examination indicates lobar consolidation, bronchial breath sounds, increased fremitus, and dullness to percussion. Pleural effusion and empyema are likely present as well.

Treatment

Patients who have community-acquired Klebsiella pneumonia are usually in respiratory distress. Consequently, mechanical ventilation is usually indicated. Because pneumonia is a shunt-producing disease, PEEP and elevated FIO₂s are often used to correct refractory hypoxemia.

The following forms of bronchial hygiene therapy are sometimes useful: postural drainage with percussion and/or vibration, high frequency compression/oscillation therapy, flutter valve, intra-pulmonary percussive ventilation, and positive expiratory pressure therapy. Pneumonia patients who do not have significant sputum production will not benefit from bronchial hygiene.

Sputum and blood must be obtained for C&S to identify the offending microbe. Third-generation cephalosporins such as cefotaxime (Claforan) and ceftriaxone (Rocephin) are used to treat Klebsiella pneumonia. Claforan inhibits bacterial growth by disrupting bacterial cell wall synthesis. Rocephin inhibits bacterial growth by binding to one or more penicillin-binding proteins. Carbapenems, aminoglycosides, and quinolones are other medications used to treat Klebsiella infections. These drugs are often used as monotherapy or in combination with each other, and should be given for at least 14 days.

Klebsiella and other gram-negative bacteria are notorious for their ability to produce beta-lactamases, which are enzymes that render penicillin and some other antibiotics useless. Penicillin and penicillin derivatives (e.g., cephalosporins) are beta-lactam antibiotics, which produce their bactericidal effect by inhibition of cell wall synthesis. Beta-lactamases are the response of bacterial adaptation to adverse factors (i.e., antibiotics) in the bacteria's environment.

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Other common hospital-acquired infections caused by Klebsiella are urinary tract infections, surgical wound infections, and infections of the blood. These can progress to shock and death if not treated early and aggressively.

Prognosis

Regardless of the treatment, community-acquired Klebsiella pneumonia has a 50% mortality rate. Because K. pneumoniae causes a necrotizing pneumonia, patients who survive often have a scarred, shrunken upper lobe containing several cavities, which resembles chronic cavitory tuberculosis.

Because the transmission of Klebsiella microbes is via the gastrointestinal tracts and hands of health care personnel, strict handwashing and patient isolation are effective means of controlling the spread of Klebsiella infections.