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Short Communication

Monitoring MDR Evolution and Transmission Dynamics of MDR Klebsiella Pneumonia

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Abstract

A Gram-negative, non-motile, capsuled, lactose fermenting, facultatively anaerobic, rod shaped bacteria called Klebsiella pneumoniae. On MacConkey agar, it manifests as a mucoid lactose fermenter. Although it is a natural component of the flora in the mouth, skin, and intestines [1], it can harm the lungs of both humans and animals if aspirated (inhaled), notably the alveoli (in the lungs), resulting in bloody sputum.

It is the most important member of the Enterobacteriaceae family's Klebsiella genus when used in a therapeutic setting. Clinical samples from humans have also shown the presence of K. oxytoca and K. rhinoscleromatis. Klebsiella species have recently emerged as significant nosocomial infection pathogens. About 30% of the strains can fix nitrogen under anaerobic conditions, and they are found in soil where it naturally occurs [2].

As a free-living diazotroph, K. pneumoniae's nitrogen fixation mechanism has undergone extensive research and is of agricultural importance because it has been shown to boost crop yields in agricultural settings [3].

On their cell surfaces, members of the Klebsiella genus normally express two different types of antigens. The Lipopolysaccharide (LPS), which comes in nine different types, contains the first antigen, called O antigen. The second is K antigen, an over 80 variety capsular polysaccharide [4]. Both support serogrouping and contribute to pathogenicity.

It has a close relationship to K. oxytoca, which it differs from in that it is indole-negative and can grow on melezitose but not 3-hydroxybutyrate.

Clinical Significance

In general, immune system compromised individuals are more likely to get Klebsiella infections. Debilitating illnesses most frequently strike middle-aged and older males.

This patient group, which includes people with diabetes, alcoholism, cancer, liver illness, chronic obstructive lung disorders, glucocorticoid medication, renal failure, and some occupational exposures, is thought to have compromised respiratory host defences (such as papermill workers). When a person is in the hospital for another cause, they frequently contract these diseases (a nosocomial infection).

Following contact with contaminated tools, faeces are the main cause of patient infection.

The most frequent illness brought on by Klebsiella bacteria outside of a hospital is pneumonia, most frequently seen as bronchopneumonia and bronchitis. These patients are more likely to experience pleural adhesions, lung abscess, cavitation, and empyema.

Even with antibiotic therapy, it has a fatality rate of about 50%. For those who also have bacteremia, the death rate can be very close to 100%. In addition to pneumonia, Klebsiella can also infect the lower biliary system, surgical wound sites, and the urinary tract.

Pneumonia, thrombophlebitis, urinary tract infection, cholecystitis, diarrhoea, upper respiratory tract infection, wound in-

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fection, osteomyelitis, meningitis, bacteraemia, and septicemia are among the list of clinical disorders.

Contamination of the device poses a risk for patients who have invasive devices in their bodies, such as those used in neonatal wards, respiratory support devices, and urinary catheters.

The likelihood of nosocomial infection with Klebsiella bacteria can also be influenced by the use of antibiotics. The bacterial infiltration into the circulation can be followed by sepsis and septic shock.

Ankylosing spondylitis may be brought on by HLA-B27 mimicry with two Klebsiella surface molecules, according to research from King's College, London [5]. In older adults with urinary tract infections, Klebsiella comes in second place to E. coli.

Additionally, it is an opportunistic infection for people who have intestinal pathogenicity, rhinoscleroma, nasal mucosa atrophy, and chronic pulmonary illness. K. pneumoniae is developing new strains that are resistant to antibiotics [6].

Evolution of Multi Drug resistance (MDR) Strains:

Numerous antibiotics are frequently ineffective against Klebsiella species. As of right now, plasmids are thought to be the main source of the resistance genes [7]. Klebsiella is resistant to a wide range of antibiotic classes if it can manufacture Extended-Spectrum Beta-Lactamases (ESBL).

The most prevalent ones are resistance to tetracyclines, chloramphenicol, aminoglycosides, fluoroquinolones, and trimethoprim/sulfamethoxazole [8]. Infection by Carbapenem-Resistant Enterobacteriaceae (CRE) or Enterobacteriaceae that produce carbapenemase is becoming a significant problem in healthcare settings [9]. Carbapenem-Resistant Klebsiella Pneumoniae is one of numerous CREs (CRKP).

There has been a steady rise in CRKP over the past ten years, but the outbreak of this newly developing nosocomial disease that started in Israel's healthcare system in 2006 is perhaps what made it so well-known [10]. It was initially mentioned in the USA in North Carolina in 1996: [11] since then, CRKP has been found in 41 states; [12] and it is regularly seen in some hospitals in New York and New Jersey.

It is currently the CRE species that Americans encounter most frequently. CRKP is resistant to practically all antimicrobial medicines now on the market, and infections with CRKP have caused high rates of morbidity and mortality, especially in those who have spent a long time in the hospital, are seriously ill, and have been exposed to invasive devices (e.g., ventilators or central venous catheters). The issue is that when dealing with resistant bacterial strains, carbapenem is frequently utilised as a last resort medication.

Healthcare practitioners may be unable to do much, if anything, to treat patients with infections caused by new, minor mutations. The Enterobacteriaceae are susceptible to carbapenem resistance due to a variety of reasons. They include the hyperproduction of CTX-M extended-spectrum beta-lactamase with a porin mutation or drug efflux, ampC beta-lactamase with an outer membrane mutation, and carbapenemase production. The synthesis of the carbapenemase enzyme blakpc by CRKP is the main method of resistance.

The probability of transmission is increased since the gene encoding the blakpc enzyme is located on a mobile genetic component (a transposon; the particular transposon in question is known as Tn4401). Because some blakpc carrying bacteria have minimum inhibitory concentrations that are high but still fall within the carbapenems' sensitive range, CRE can be challenging to detect.

These strains are not recognised by conventional susceptibility testing standards as possible clinical or infection control hazards since they are responsive to carbapenems. During nosocomial epidemics, patients with undiagnosed CRKP colonisation have served as transmission reservoirs. The recommended course of action has altered as the organism has acquired resistances, as is the case with many bacteria. The portion of the body that is infected and local susceptibility patterns determine which antimicrobial agent or agents should be used.

For patients with serious infections, it is preferable to start them on a brief course of combination therapy (48–72 hours), and then switch them to a particular monotherapy once the patient's individual susceptibility pattern is understood.

The medications used to treat such susceptible isolates of Klebsiella include ampicillin/sulbactam, piperacillin/tazobactam, ticarcillin/clavulanate, ceftazidime, cefepime, levofloxacin, norfloxacin, gatifloxacin, moxifloxacin, meropenem, and ertapenem if the specific Klebsiella in a Meropenem has been suggested by several specialists for usage in patients with Klebsiella that produces ESBL. Meropenem is said to produce the best bacterial clearance, according to the claim. Antibiotic usage is frequently insufficient. After the patient begins taking antimicrobial medications, surgical clearance (typically performed as interventional radiology drainage) is commonly required.

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