Case Report

The Portrayal of Microbes in Respiratory Medicine

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Abstract

Respiratory diseases caused by a number of infectious agents including *Streptococcal pneumonia, streptococcal pyogenes, Klebsiella pneumonia H. influenzae, Legionella pneumophilia, Mycoplasma pneumonia, Coxiella Bunetii, chlamydia pisittaci.* These microbes enter into the lungs and cause primary cases of pneumonia. The whole respiratory epithelium down to the internal bronchioles is ciliated. The cilia and the thin film of mucus covering them have the critical function of trapping foreign particles, including bacteria, and propelling them toward the pharynx. They contribute to the prevention of respiratory infection as they do the macrophages utilizing their secretory, phagocytic, and bactericidal activity. Despite the wonderful defense, the bacterium escapes and settles in the lungs to produce diseases. Pneumonia is the term used to describe inflammation of the lung. Pneumococcal pneumonia is characterized by homologous consolidation of one or more lobes or segments. Pneumococcal pneumonia occurs at all ages but most frequently in early and middle adult life. Pneumonia is characterized with the rise in body temperature 39-40°C, remain associated with painful cough initially dry, but later patient develops production of tenacious sputum, which is often rusty and occasionally bloodstained. Staphylococcal pneumonia is caused due to *Streptococcus aureus*, which may occur either as a primary respiratory infection or as a blood-borne infection from a staphylococcal lesion elsewhere in the body. Klebsiella pneumonia is caused due to *K. pneumoniae*, a rare disease with high mortality. There is usually massive consolidation and excavation of one or more lobes, with the upper lobes being most often involved and with large amounts of purulent sputum, sometimes characteristic red currant jelly sputum. Legionella pneumonia is caused by Gram-negative bacillus *L. pneumophila*, which is usually transmitted in water droplets from infected cisterns used to provide water for showers, particularly in warm climates. It is often a serious and occasionally a fatal illness. *C. psittaci* causes psittacosis (ornithosis), a systemic illness contracted from infected birds. Pneumonia associated with it may be extensive, with severe toxemia. *M. pneumoniae* is a pleomorphic bacterium; symptoms of mycoplasma pneumonia are mild compared to other cases of pneumonia (walking pneumonia). On the other hand, liver function test derangements and dyselectrolytemia are more common. It is susceptible to tetracyclines though a few strains are sensitive only to erythromycin.

Keywords: Atypical pneumonia, *Chlamydia psittaci*, granulocyte colony-stimulating factor, *Haemophilus influenzae*, Klebsiella pneumonia, Legionella pneumonia, *Mycoplasma pneumoniae*, pneumococcal pneumonia, staphylococcal pneumonia, tumor necrosis factor

Introduction

Pneumonia is the leading cause of death among children under 5 years of age, particularly in developing countries. According to the World Health Organization (WHO), more than 15 million cases are representing 7%–13% of annual pneumonia cases, which necessitate hospital admission due to their severity.[1] Atypical pneumonia is caused by microbes other than *Streptococcus pneumoniae, klebsiella pneumonia, Haemophilus influenzae, staphylococcus aureus,* and *Moraxella catarrhalis*. These atypical organisms include special bacteria, viruses, fungi, and protozoa.[2] Cytokines are

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released in response to the inflammatory reaction and cause the constitutional symptoms; for example, interleukin-1 (IL-1) and tumor necrosis factor (TNF) cause fever. Chemokine-like IL-8 and colony-stimulating factors such as granulocyte colony-stimulating factor (G-CSF) promote chemotaxis and neutrophils maturation, respectively, resulting in leukocytosis on serological laboratory and purulent secretions. Sometimes, even erythrocytes cross this barrier and result in hemoptysis.[3,4] Pneumococcal pneumonia caused by *S. pneumoniae*, is the most common type of primary pneumonia. It is characterized by homologous consolidation of one or more lobes or segments. *Klebsiella pneumonia* also causes pneumonia as the form of bronchopneumonia and also bronchitis .[5] In 1882, Carl Friedlander described *Klebsiella pneumoniae* for the first time. He described it as an encapsulated bacillus after isolating the bacterium from the lungs of those who died from pneumonia.[6]

Staphylococcal pneumonia secretes a series of toxins including include alpha toxin, beta toxin, delta toxin, and several bi-component toxins that act on cell membranes. Strains of *S. aureus* host phages, the prophage F-PVL that produces Panton-Valentine leukocidin (PVL), to increase virulence. The bi-component toxin PVL is associated with severe necrotizing pneumonia in children. The list of small RNAs involved in the control of bacterial virulence in *S. aureus* is growing.[7] *S. aureus* pneumonia is a common, potentially life-threatening infection caused by this human pathogen.[8] Pfeiffer's bacillus, or *Bacillus influenzae*, repeatedly occurred in pathological samples obtained from persons who died of fatal respiratory infections.[9] It is ironic that the organism, eventually designated *H. influenzae*, which was once so widely evident in laboratory tests (albeit, only fleetingly considered the cause of the "Spanish flu"), is now a rarely visible culprit that is still responsible for a considerable toll of childhood illness in resource-poor countries.[10] The incidence of invasive *H. influenzae* type B (Hib) diseases has dramatically decreased because of widespread use of the Hib conjugate vaccine. In contrast, NTHi strains have become the most common cause of invasive disease in all age groups in countries with routine Hib vaccination.[11] Many cases are diagnosed after presenting chest infections that do not respond to penicillin or first-generation cephalosporin. A chest X-ray can identify alveolar consolidation.[12] Both *H. influenzae and S. pneumoniae* can be found in the upper respiratory system of humans. In an *in vitro* study of competition, *S. pneumoniae* always overpowered *H. influenzae* by attacking it with hydrogen peroxide and stripping off the surface molecules *H. influenzae* needs for survival.[13]

Unencapsulated *H. influenzae* is often observed in the airways of patients with chronic obstructive pulmonary disease (COPD).[14] Members of the genus *Haemophilus* will not grow on blood agar plates, as all species require at least one of these blood factors for growth: hemin (X-factor) and/or nicotinamide adenine dinucleotide (V-factor).[15] *Legionella* is a genus of pathogenic Gram-negative bacteria that include the species *L. pneumophila*, causing legionellosis all illnesses caused by *Legionella*, including a pneumonia-type illness called Legionnaires' disease and a mild flu-like illness called Pontiac fever.[16] *Legionella* transmission occurs via inhalation of water droplets from contaminated sources that allow the organism to grow and spread.[13] Chest radiographs (X-ray photographs) often show a pulmonary infection before physical signs of atypical pneumonia are observable at all. This is occult pneumonia. It has been reported that occult pneumonia often present in patients with pneumonia caused by *Streptococcus pneumonia*.[14] To minimize the burden of severe pneumonia, the efforts aiming to identify the causative agents are mandatory. Blood, sputum, fluids, or tissue samples from the lungs for culture are usually useful, however not promising in young children due to difficulty or inadequacy in the collection of samples, as well as due to transportation difficulties or lack of technical facilities in developing countries.[15] Blood culture is necessary for appropriate management of clinically severe pneumonia in children under 5 years of age.[7]

Historical Perspectives of Various Microbes

The genus *Klebsiella* was named after the German microbiologist Edwin Klebs (1834–1913). It is also known as Friedlander's *Bacillus* in honor of Carl Friedlander, a German pathologist, who proposed that this bacterium was the etiological factor for the pneumonia seen, especially in immunocompromised individuals such as people with chronic diseases or alcoholics. Community-acquired pneumonia (CAP) caused by *K. pneumoniae* may be called Friedlander's *Bacillus*.[17] German scientist Friedrich Julius Rosenbach identified *S. aureus*, discriminating and separating it from *Staphylococcus albus*, a related bacterium.[18] A study by Fitzgerald *et al*. revealed that approximately 22% of the *S. aureus* genome is noncoding and thus can differ from bacterium to bacterium. This demonstrates that there is a large range of infectious ability within the species.[19] *S. aureus* different strains can secrete different enzymes or bring different antibiotic resistances to the group, increasing its pathogenic ability.[20] *H. influenzae* (formerly called Pfeiffer's *Bacillus* or *B. influenzae*) is a Gram-negative, coccobacillary, facultative anaerobic pathogenic bacterium of the family *Pasteurellaceae*. *H. influenzae* was first described in 1892 by Richard Pfeiffer during an influenza pandemic.[21]

In 1898, Nocard and Roux were the first to isolate an agent assumed to be the cause of cattle pneumonia and named it "microbe de la peripneumonie."[22] Encapsulated strains were classified on the basis of their distinct capsular antigens. The six generally recognized types of encapsulated *H. influenzae* are a, b, c, d, e, and f.[23] Effective vaccines for Hib have been available since the early 1990s and are recommended for children under 5 years of age and asplenic patients. The WHO recommends a pentavalent vaccine, combining vaccines against diphtheria, tetanus, pertussis, hepatitis B, and Hib. There is not yet sufficient evidence on how effective this

Table 1; The table summarize treatment strategies for inpatients with community acquired pneumonia.

Types of Patients	Standard treatment options	Prior respiratory isolation of MRSA	Prior respiratory isolation of *P. aeruginosa*	Hospitalization and infusion of antibiotics in case of MRSA	Hospitalization and infusion of antibiotics in case of *P. aeruginosa*
Non severe inpatient pneumonia	Beta lactam along with macrolides or fluoroquinolones	Culture and confirmatory PCR for MRSA for continuation of therapy	Culture and confirmatory PCR for *P. aeruginosa* for continuation of therapy	If MRSA culture and or PCR positive extension of therapy	If *P. aeruginosa* culture and or PCR positive extension of therapy
severe inpatient pneumonia	Beta lactam along with macrolides or fluoroquinolones along with macrolides	Culture and confirmatory PCR for MRSA for continuation of therapy	Culture and confirmatory PCR for *P. aeruginosa* for continuation of therapy	If MRSA culture and or PCR positive extension of therapy	If *P. aeruginosa* culture and or PCR positive extension of therapy

pentavalent vaccine is in relation to the individual vaccines.[24] Legionnaires' disease acquired its name in July 1976, when an outbreak of pneumonia occurred among people attending a convention of the American Legion at the Bellevue-Stratford Hotel in Philadelphia. Of the 182 reported cases, mostly men, 29 died.[25] Microorganisms from other sources, having properties similar to the pleuropneumonia organism of cattle, soon came to be known as pleuropneumonia-like organisms (PPLOs), but their true nature was unknown. Many PPLOs were later proven to be the cause of pneumonias and arthritis in several lower animals.[26] *Chlamydia pneumoniae* is a species of *Chlamydia*, an obligate intracellular bacterium that infects humans and is a major cause of pneumonia. It was known as the Taiwan acute respiratory agent from the names of the two original isolates Taiwan (TW-183) and a sensitive respiratory isolate-designated AR-39. Briefly, it was known as *Chlamydophila pneumoniae*, and that name was used as an alternate in some sources.[27]

Research on the Development of Lower Respiratory Tract Microbes

The exact starting time point for the bacterial colonization of the human body cannot be accurately determined. Until recently, amniotic fluid, which fills fetal lungs prenatally, was considered sterile.[28] Detectable microbial communities in the multiple body sites have been identified in newborns as early as <5 min after delivery, and their synthesis initially resembles the maternal vagina or skin microbiota composition, depending on the mode of delivery (vaginal or cesarean section).[29] This premature microbiome has been shown to change in design and diversity and mature functionally during the first 2–3 years of life, after which it gradually stabilizes to a pattern closely matching that of adults.[30] Bacterial lung infections are usually categorized as acute or chronic depending upon the rate at which they evolve, but more likely related to the quality they resolve after antibiotic therapy.[31] This approach applies to COPD, where routine culturing is not recommended.[31]

Diagnosis of "Lower Respiratory Tract Bacterial Infection"

On initial investigations to confirm Lower respiratory tract infection (LRTI), the severity of LRTI includes complete blood picture, renal function tests, liver function tests, arterial blood gas analysis, and serum electrolytes, and imaging studies include chest X-ray for the localization of the infected foci, which can reveal lobar pneumonia or bronchopneumonia [Figure 1]. To identify the etiology of pneumonia, sputum cultures, blood cultures and urine cultures (if suspecting pneumonia secondary to bacteremia or urinary tract infection) are essential. If the patient is not able to cough out sputum, to evaluate the cause of pneumonia-bronchoscopy can be performed and Bronchoalveolar lavage (BAL) from the localized segment or lobe will be sent for cultures that can give etiology for specific antibiotic management. Mini-BAL can be sent for the cultures for the etiological diagnosis. Bronchial and pulmonary secretions or exudates are often studied by examining sputum. The most misleading aspect of sputum examination is almost inevitable contamination with saliva and mouth flora. Thus, finding *Candida*, *S. aureus*, *or even S. pneumonia* in the sputum of a patient with pneumonitis has no etiologic significance unless supported by the clinical picture. Meaningful sputum specimens should be expectorated from the lower respiratory tract and should be grossly distinct from saliva. The presence of many squamous epithelial cells suggests heavy contamination with saliva. A large number of polymorphonuclear (PMN) leukocytes suggest a purulent exudate.

Sputum may be induced by the inhalation of heated hypertonic saline aerosol for several minutes. In pneumonia accompanied by the pleural fluid, the pleural fluid may yield the causative organisms more reliably than does sputum. Most community-acquired bacterial pneumonia is caused by pneumococci. In suspected tuberculosis or fungal infection, gastric washings may yield organisms when expectorated materials fall to do so. Specimens obtained by mini BAL or bronchoscopy or TBLB or percutaneous or open lung biopsy

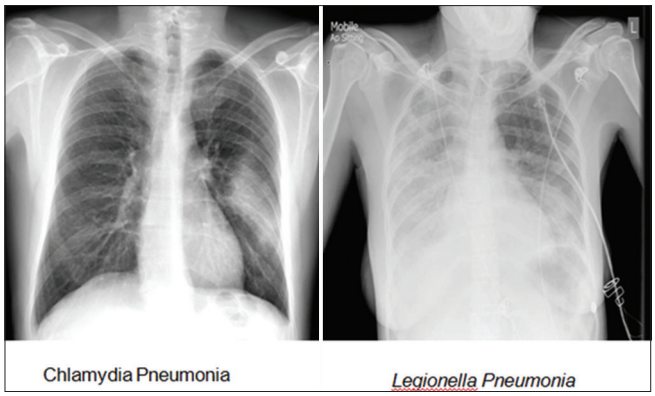

Figure 1: X-ray image of chest and cause of pneumonia by *Chlamydia* pneumonia and *Legionella* pneumonia

are necessary in the diagnosis of Pneumocystis pneumonia or infection due to *Legionella* or other organisms. Smears of purulent flecks or granules from the sputum stained by Gram stain or acid-fast methods may reveal causative organisms and PMN leukocytes. Specimens obtained by bronchoscopy and lung biopsy should also be cultured on other media (e.g., for anaerobes and *Legionella* and others).[28] In the lower respiratory tract, there is often a primary infection by viruses, e.g., adenoviruses, respiratory syncytial virus, herpes virus, and picornavirus, and thereafter, there is a secondary infection by pathogenic microorganisms from the upper respiratory tract and by spread through the blood and lymph channels; more rarely, there is a direct extension of disease from other affected tissues, such as the liver. The mucus membrane of this part of the respiratory tract is probably sterile in health, but direct examination involves bronchoscopy or lung biopsy, neither of which is indicated in the ordinary course of diagnosis of infection. Specimens obtained by bronchoscopy or open lung biopsy may be necessary for the diagnosis of pneumocystis pneumonia.[32] Atypical pneumonias caused by *S. pneumoniae, H. influenzae,* or *M. catarrhalis* are typical CAPs with clinical and laboratory findings limited to the lungs.[33]

Treatment

The treatment of pneumonia is involving a precise and early clinical diagnosis; cause of pathogen and severity of disease. Here, several critical biochemical parameters are part of clinical examination before option a therapeutic intervention including age, blood pressure, respiration rate, etc. The clinical outcome allows ranking patients based on severity of disease. A score of 0 or 1 usually associated with low mortality(1.5%) can be treated at home, score two associated with intermediate mortality (9.2%) options for treatment include short-stay inpatient/hospital supervised outpatient and score three or more associated with high mortality (22%) treated at ICU.

Combination therapy

B amoxicillin/clavulanate 500 mg/125 mg three times daily, or amoxicillin/clavulanate 875 mg/125 mg twice daily, or 2000 mg/125 mg twice daily, or a cephalosporin (cefpodoxime 200 mg twice daily or cefuroxime 500 mg twice daily); and macrolide (azithromycin 500 mg on the 1st day then 250 mg daily, clarithromycin [500 mg twice daily or extended-release 1000 mg once daily]) or doxycycline 100 mg twice daily OR monotherapy with a respiratory fluoroquinolone (levofloxacin 750 mg daily, moxifloxacin 400 mg daily, or gemifloxacin 320 mg daily) are prescribed [Table 1]. In inpatient setting, antibiotic regimens recommended for empiric treatment of CAP in adults without risk factors for methicillin-resistant *S. aureus* and *Pseudomonas aeruginosa*-nonsevere CAP – in no order of preference – are combination therapy with a b-lactam (ampicillin 1 sulbactam 1.5–3 g every 6 h, cefotaxime 1–2 g every 8 h, ceftriaxone 1–2 g daily, or ceftaroline 600 mg every 12 h) and a macrolide (azithromycin 500 mg daily or clarithromycin 500 mg twice daily) or monotherapy with a respiratory fluoroquinolone (levofloxacin 750 mg daily and moxifloxacin 400 mg daily).

The third option for adults with CAP who have contraindications to both macrolides and fluoroquinolones is the combination therapy with a b-lactam (ampicillin 1 sulbactam, cefotaxime, ceftaroline, or ceftriaxone, doses as above) and doxycycline 100 mg twice daily. There is growing consensus in case of Inpatient Setting; Should Patients with Suspected Aspiration Pneumonia (ATS/IDSA) not routinely recommend adding anaerobic coverage for suspected aspiration pneumonia unless lung abscess or emphysema is suspected?. Patients who aspirate gastric contents are considered to have aspiration pneumonitis. Many of these patients have a resolution of symptoms within 24–48 h and require only supportive treatment without antibiotics. Studies evaluating the microbiology of patients with aspiration pneumonia in the 1970s showed high rates of isolation of anaerobic organisms; however, these studies often used transtracheal sampling and evaluated patients late in their disease course, two factors that may have contributed to a higher likelihood of identifying anaerobic organisms. Several studies of acute aspiration events in hospitalized patients have suggested that anaerobic bacteria do not play a significant role in etiology. With increasing rates of *Clostridium difficile* infections (frequently associated with the use of clindamycin), the question of adding empiric anaerobic coverage (clindamycin or b-lactam/b-lactamase inhibitors) in addition to routine CAP treatment in patients with suspected aspiration is an important one.

Corticosteroids are indicated in patients with CAP and refractory septic shock. In adults with CAP who test positive for influenza, treatment regimen including antiviral therapy-anti-influenza treatment, such as oseltamivir, is prescribed for adults with CAP, in the inpatient setting as well as in the OP setting, independent of the duration of illness before diagnosis. Treatment within 2 days of symptom onset or hospitalization may result in the best outcomes, although there may be benefits up to 4 or 5 days after symptoms begin. The use of anti-influenza agents in the OP setting reduces the duration of symptoms and the likelihood of lower respiratory tract

complications among patients with influenza, with most benefit if therapy is received within 48 h after onset of symptoms. The ATS/IDSA recommends that standard antibacterial treatment be initially prescribed for adults with clinical and radiographic evidence of CAP who test positive for influenza in the inpatient and OP settings. *Staphylococcus aureus*; A combination therapy with penicillinase-resistant penicillin or a cephalosporin (in case the organism is methicillin-sensitive *S aureus* [MSSA]) and clindamycin or fluorquinolone.[34] *Klebsiella pneumonia*; A third-generation cephalosporin, carbapenems, amino glycosides, and quinolones.

Atypical Pneumonia

- *L. pneumophila*: The treatment should have high intracellular concentrations such as macrolide, quinolones, keto lives, tetracyclines, and rifampins
- *M. pneumonia*: The drugs of choice are either azithromycin or clindamycin
- *C. pneumoniae*: Tetracyclines and macrolide are the drugs of choice
- C. *psittaci*: Tetracycline or doxycycline is the drug of choice. Azythromycin should be considered as the second line of defense
- *H. influenzae*: Intravenous (IV) third-generation cephalosporin is the drug of choice until antibiotic sensitivities become available of intramuscular ceftriaxone when IV administration is not available
- *S. aureus*: combination therapy with penicillinase-resistant penicillin or a cephalosporin (in case the organism is MSSA) and clindamycin or fluoroquinolones is the drug of choice[34]
- *K. pneumoniae:* Third-generation cephalosporins, carbapenems, aminoglycosides, and quinolones are the drugs of choice[35]
- *M pneumoniae*: the drugs of choice are either azithromycin or clindamycin
- *C. pneumoniae*: tetracyclines and macrolide are the drugs of choice
- *C. psittaci*: Tetracycline or doxycycline is the drug of choice. Azithromycin should be considered as the second line of defense.

Why is the problem significant in research?

Acute and chronic respiratory tract infections are a common cause of inappropriate antimicrobial prescriptions. Antimicrobial therapy leads to the development of resistance and opportunistic pathogens that substitute the indigenous microbiota. Staphylococci, in general, are sensitive to many antibiotics, such as benzylpenicillin, cloxacillin, cephalosporin, tetracyclines, chloramphenicol, erythromycin, fucidin, clindamycin, vancomycin, streptomycin, and gentamycin. Still, strains from different patients and carriers differ in the pattern and degree of their sensitivity to the various drugs, and many strains are now resistant to some of the drugs at the highest concentrations that can safely be achieved in the patient's tissues. Staphylococci are poorly susceptible to sulfonamides except when sulfonamides are used in combination with trimethoprim, as in the preparation of cotrimoxazole. Penicillin-resistant strains of *S. aureus* existed before the introduction of penicillin into medical use in 1942. Still, they were rare and comprised only a tiny proportion (less than 1%) of the strains isolated at that time.

The property of penicillinase production may originally have evolved because it acted on some natural substrate chemically, similar to penicillin, or because it protected the cocci when growing in habitats contaminated with penicillin-like antibiotic, e.g., fungal skin lesions. After the medical use of penicillin had become general, there was a progressive, massive increase in the prevalence of the penicillin-resistant staphylococci. The growth was more rapid in hospitals than in the general community. Research program for the next-generation world imaging plays a crucial role in lung infections. A computed tomography scan must be carried out when there is a strong clinical suspicion of pneumonia accompanied by standard, ambiguous, or nonspecific radiography. This scenario occurs most commonly in immunocompromised patients.[36] Significant challenges remain in accurately defining the potential role of bacteria in the inflammatory process and how best to optimize the use of antibiotics without the overuse of this limited resource.[37]

Present key findings with respect to central research questions

Pneumonia is the term used to describe inflammation of the lung. There are many different kinds of pneumonia: some may be common and some may be rare. Primary pneumonia, caused by *S. pneumoniae*, is the most common type of primary pneumonia. Other bacteria that may cause primary pneumonia include *S. pyogenes, K. pneumoniae, H. influenzae,* and *L. pneumophila* and small bacteria such as *M. pneumoniae, C. burnetii,* and *C. psittaci.* Anaerobic organisms that include *A israelii* are rare causes of primary pneumonia. In secondary pneumonia, *H. influenzae* and some types of *S. pneumoniae* and sure of the bacteria forming the flora of the upper respiratory tract and mouth are the organisms most frequently cultured from sputum. Pneumococcal pneumonia is characterized by the consolidation of one or more lobes. The disease occurs at all ages but most often in the early and middle adult of life. The highest incidence is in winter.

Research on the Development of Lower Respiratory Tract Microbe

Although not explicitly studied in humans, the development of the lung microbiome probably adheres to that of the rest of the human body microbial ecosystem. Until recently, amniotic fluid, which fills fetal lungs prenatally, was considered sterile. This historical belief was challenged by discovering bacterial DNA in the amniotic fluid and placental samples.[38] Detectable microbial communities in multiple body sites have been identified in newborns as early as < 5 min after delivery, and their synthesis

initially resembles the maternal vagina or skin microbiota composition, depending on the mode of delivery (vaginal or cesarean section).[39] This premature microbiome has been shown to change in design and diversity and mature functionally during the first 2–3 years of life, after which it gradually stabilizes to a pattern closely matching that of adults.[40] Persistent lung infections and bacterial lung infections are usually categorized as acute or chronic, depending upon the rate at which they evolve. Still, more likely related to the quality, they resolve after antibiotic therapy.[41] Most acute lung infections do uphold Koch's postulates very well. Sputum cultures and staining techniques, when appropriately collected and culture positive, are usually helpful in determining which antibiotics to use.[42]

Perspectives of Future Research Work

It is expected that future research in bacterial gene expressions, metagenomics, longitudinal analysis, and host-microbiome animal models will help move toward targeted microbiome interventions in respiratory diseases. In hospitals, the areas at highest risk for severe staphylococcal infections are newborn nurseries, ICUs, operation rooms, and cancer chemotherapy wards. The massive introduction of "epidemic" pathogenic *S. aureus* into these areas may lead to severe clinical disease. Personnel with active staphylococcal lesions and carriers may have to be excluded from these areas. In such individuals, the application of topical antiseptics (e.g., chlorhexidine or bacitracin cream) to nasal or perineal carriage sites may diminish the shedding of dangerous organisms. Antiseptics such as hexachlorophene used on the skin of newborns decrease colonization by staphylococci, but toxicity prevents their widespread use. Encapsulated Hib is transmitted from person to person by the respiratory route. Because an increase in the number of adults lacks bactericidal antibody and is susceptible to systemic *Haemophilus* infections, immunization with capsular polysaccharides (PRP vaccine) is now proposed to mothers at high risk who lack antibody. It should also be given to all children over age 2 years and to those between 18 and 23 months of age who are at increased risk. *K. pneumonia* is present in the respiratory tract and feces of about 5% of normal individuals. *K. pneumonia* can produce extensive hemorrhagic necrotizing consolidation of the lung. *L. pneumophila* is ubiquitous in the environment.[43]

They are present in soil and freshwater lakes and streams and have been found air-conditioning systems and washing facilities, e.g., shower stalls. The later sources have been responsible for outbreaks of human disease, especially in hospitals. Chlorination and heating of water and cleaning can help control the multiplication of *Legionella* in water and air conditioning systems. Legionellae are not communicable from infected patients to others. Isolation of infected livestock will prevent the highly contagious pleuropneumonias. No vaccines are available. Mycoplasma pneumonia behaves like a communicable viral respiratory disease. *C. psittaci* causes psittacosis, and the disease acquired to humans from contact with birds and also infection of psittacine birds (parrots, parakeets). Birds kept as pets have been an essential source of the human condition. Latent infections often flared up in these birds during transport and crowding. Sick birds excrete large quantities of the infectious agent.

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Conflicts of interest

There are no conflicts of interest.

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