Review
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A Review: Hospital Acquired Infection in some Gram Negative Pathogenic Bacterial Strains

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Abstract

Objective: This review aims to update healthcare workers on the current scientific understanding of hospital-acquired infections, with a focus on describing the pathophysiology and patterns of antimicrobial resistance, particularly concerning *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Klebsiella pneumoniae*.

Methods: Data on hospital-acquired infections were collected globally, with a specific emphasis on the Eastern Mediterranean, South-East Asia, Europe, and the Western Pacific regions. Infection rates, predominant pathogens, and antimicrobial resistance patterns were analyzed to provide insights into the current landscape of nosocomial infections.

Results: Hospitals in the Eastern Mediterranean and South-East Asia regions reported the highest rates of nosocomial infections (11.8% and 10.0%, respectively), while rates in Europe and the Western Pacific were 7.7% and 9.0%, respectively. Infections typically arise from invasive medical equipment and surgical operations, with lower respiratory tract and bloodstream infections being particularly hazardous. Gram-negative bacterial infections, notably, exhibit worrisome antibiotic resistance patterns, potentially developing multiple mechanisms against various antibiotics.

Conclusion: The emergence of antimicrobial resistance presents a significant threat to patient safety, compounded by challenges in discovering new antibiotics. Factors such as high costs and lengthy drug development processes contribute to this concern. Healthcare workers must remain abreast of evolving antimicrobial resistance patterns, especially concerning pathogens like *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Klebsiella pneumoniae*, to implement effective infection control measures and preserve the efficacy of existing antimicrobial agents.

Keywords: Hospital acquired infection, bacteria, pathogens, antibiotics, CDC

Introduction

An infection obtained at a hospital by a patient who was admitted for a reason other than the infection is known as a nosocomial infection or a "hospital acquired infection." These infections are most often acquired during hospitalisation and present 48 hours after admission.[1] The one where the disease was not present or incubating upon admission to a hospital or other health care facility. Includes infections acquired in the hospital but manifesting after discharge and occupational infections among the facility's personnel. Worldwide, nosocomial infections impact both wealthy and resource-poor countries equally.[2] When patients become sick while receiving medical treatment, they are more likely to be a victim and have a worse outcome. Hospitals in the Eastern Mediterranean and South-East Asia regions had the highest rates of nosocomial infections (11.8% and 10.0%), while in Europe and the Western Pacific, the rates were 7.7 and 9.0 percent, respectively.[3] A patient's functional incapacity and emotional stress are exacerbated by hospital-acquired infections, which can lead to debilitating conditions that impair quality of life. Another significant cause of death is nosocomial infections. Nosocomial infections can be caused by a wide range of bacteria, viruses, fungi, and parasites. Hospital infections may be caused by an infected person's microorganism (cross-infection) or may be caused by an infection in the patient's own micro flora (endogenous infection). An inanimate object or substance recently infected by another human source may contain some microbes.[4]

Epidemiology

It is important to understand epidemiology because it's the study of how disease spreads from person to person and from population to population. Epidemiology also includes the study of how populations respond to outbreaks of disease, identifying endemic/enzootic and epidemic/epizootic infections, detecting multiple strains in populations and/or individuals, and addressing alternative epid subtyping by molecular means has been found superior to phenotypic methods in most cases because to its greater discrimination and less dependence on organism responses to environmental signals.[5]

Bacterial infections are very common in cirrhotic individuals and have been associated to the development of comorbidities as well as a high short-term mortality rate. Bacterial infections in cirrhosis have a clinically significant negative impact regardless of the stage of liver disease.[6] Healthcare-associated infections were the subject of a multistate point prevalence survey conducted by the CDC in 2014 on 11,282 people from 183 US hospitals.[1] Approximately 4% of hospitalized patients, according to this data, were infected with at least one HAI. In 2011, approximately 648,000 hospitalized patients were estimated to have had 721,800 infections. Pneumonia (21.8%), surgical site infections (21.8%), gastrointestinal infections (17.1%), urinary tract infections or UTIs (12.9%), and primary bloodstream infections are the most common infections (in descending order) (9.9%, and include Catheter-associated bloodstream infections). C. difficile is the most common pathogen responsible for HAI, followed closely by

Staphylococcus aureus (10.7%), *Klebsiella* (9.9%), and *Escherichia coli* (11.1%). *Staph aureus* infections of the skin and surgical sites are common, and they can include methicillin-resistant *Staph aureus*.[1-7] When infection tracking and infection control programs are combined, infection rates can be reduced by a third, according to the Study on the Effectiveness of Nosocomial Infection Control study. There has been a decrease in the occurrence of certain HAIs as a result of increased public awareness and strong prevention measures implemented in hospitals. HAI prevention has seen some progress as a result of rigorous infection surveillance and prevention procedures being implemented. CLABSI rates dropped by 46% between 2008 and 2013, according to the CDC.[1-8]

On May 11, 2017, a point prevalence survey was performed among inpatients at six Saudi Arabian hospitals. The total occurrence of points was 6.8 percent (114 of 1,666). Pneumonia (27.2 percent), urinary tract infections (20.2%), and bloodstream infections were the most prevalent forms of infections (10.5%). Around 19.2% of healthcare-associated infections were caused by medical devices.[9]

Pathophysiology

Mechanical ventilation, invasive medical devices, and surgical procedures are the most common causes of hospital-acquired infections. More than 30% of hospital-acquired infections are caused by Gram-negative bacteria, which are also the most common cause of hospital-acquired pneumonia.[10] Because of this, they are extremely effective at increasing or acquiring mechanisms of antibiotic drug resistance. Because their cell walls differ, gram-negative and gram-positive bacteria stain differently. They can also lead to a variety of infections, which can be treated with various antibiotics. A capsule surrounds Gram-negative bacterium. This supplement helps to keep germs out of white blood cells, which fight infection. Gram-negative bacteria are protected against antibiotics like penicillin by an outer membrane under the capsule. Endotoxins are harmful chemicals that are released when the cell membrane is damaged. Gram-negative bacterial infections are made more painful by endotoxins. Gram-negative bacterial infections are particularly dangerous because of several characteristics. Especially in the face of antibiotic selection pressure, these organisms are particularly efficient at up-regulating or acquiring genes that code for mechanisms of antibiotic drug resistance.[10] More than 30% of hospital-acquired infections are caused by gram-negative bacteria, according to previous data from the National Healthcare Safety Network in the United States. Gram-negative bacteria predominate in ventilator-associated pneumonia (47% of cases) and urinary tract infections (45%).[11] About 70% of these infections occur in American intensive care units (ICUs), according to research conducted by the American Society of Infectious Diseases.[11,12] Despite new treatments in the market, the development of resistance among pathogenic microorganisms has been increasing, particularly in patients who have been exposed to drugs for a long time. Antimicrobial medications often operate on microorganisms by constraining a metabolic path such as nucleotide synthesis, which prevents DNA and RNA synthesis, subsequent protein synthesis, and cell membrane rupture, or by competing with the substrate of an enzyme in the cell wall formation (for example, chitin synthase).[13] Microorganisms have developed a variety of strategies to defeat the medications' effectiveness, allowing them to survive drug exposure (Figure 1).

Developing countries are disproportionately affected by antimicrobial resistance (AMR) due to high rates of infectious disease, weak health infrastructure, and poor hygiene. A high percentage of hospital-acquired infections in underdeveloped countries is concerning because it affects treatment results and the development of antimicrobial resistance (AMR). If current AMR circumstances continue unchecked, by 2050 they will be the cause of almost 10 million deaths.[14] Because of their great propensity to spread among patients, pathogens such *Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa*, and *Enterobacteriaceae* are the most prevalent ones that develop antimicrobial resistance (AMR). *A. baumannii* is a particularly difficult bacterial disease to combat due to its very high level of antibiotic resistance. *Acinetobacter* is a genus of Gram-negative

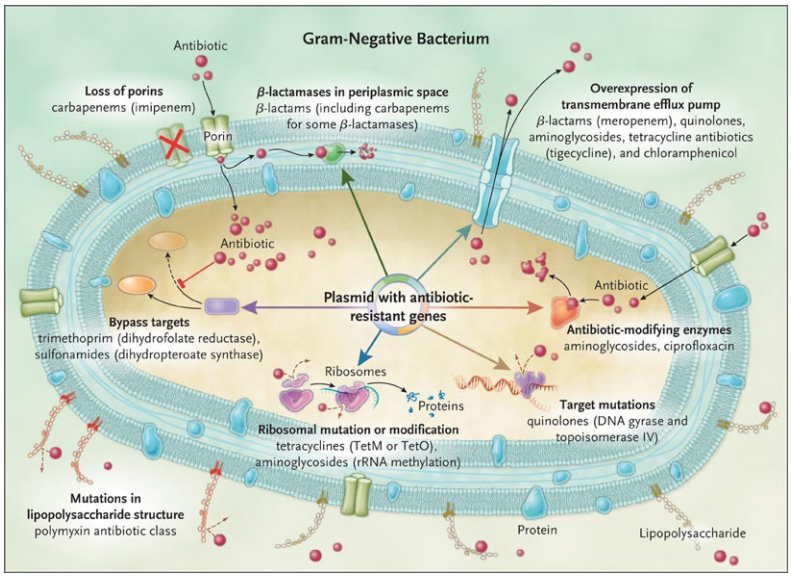

Fig. 1 **Gram-negative bacteria resistance mechanisms and antibiotics affected.**[10]

coccobacilli that does not digest lactose, is catalase-positive, is nonmotile, is nonfastidious, and does not produce oxidase.[15] Nosocomial infections are associated with *A. baumannii,* and it is intrinsically resistant to a wide range of antibiotic classes, with a high likelihood of gaining resistance. Because of *Acinetobacter* remarkable capacity to withstand desiccation, inanimate things can harbour it for months, making it easier for it to spread throughout the hospital. Hospitalizations, morbidity, and mortality have all been associated to multidrug-resistant *Acinetobacter* sp.[16]

Antimicrobial-resistant bacteria have become more prevalent due to the ongoing overuse and reckless application of antibiotics. Because of their inherent resistance to a wide range of medications and ability to produce elevated levels of multidrug resistance, bacteria such as *P. aeruginosa*, *A. baumannii*, and the *K. pneumoniae* species are receiving an increasing amount of clinical attention. Perhaps unsurprisingly, the presence of broad-specific efflux systems, which export and provide resistance to various antimicrobials, is a major contributor to this resistance's development. The principal multidrug efflux systems responsible for intrinsic and acquired MDR in the aforementioned organisms make use of a drug-proton antiporter from the RND family and have been classified into five families of antimicrobial efflux systems.[17,18]

Since treatment options for antibiotic-resistant infections are limited, the microbes that produce them appear biologically fit and capable of inflicting life-threatening infections. Drug-resistant infections are becoming more common at a time when research and development of new anti-infective drugs is decreasing substantially. This is concerning. As a result, there is concern that we may be confronted with an increasing number of infections that are untreatable in the not-too-distant future. Antibiotic-resistant bacteria have been implicated in a number of hospital-acquired illnesses in the last decade. MDR microorganisms are those that are resistant to a wide range of antimicrobial agents. Antibiotics function by interfering with the development of certain bacterial cell walls, proteins, or nucleic acids in bacteria. The antibiotic must be able to reach and bind to the bacterial target site in order to achieve this. Resistant microorganisms have a variety of mechanisms for dealing with antibiotics, some of which are enzymatic in nature, some of which involve structural alterations to antibiotic target sites, and some of which prevent an adequate concentration of the antimicrobial agent from reaching the active site.[19]

Pseudomonas aeruginosa

As a prevalent nosocomial pathogen that causes life-threatening infections, *Pseudomonas aeruginosa* should be avoided at all costs. Both growing multidrug resistance in hospital settings and the organism's inherent high tolerance to a variety of antimicrobials are factors complicating antipseudomonal chemotherapy. Multidrug resistance has been linked to greater morbidity and mortality, longer hospital stays, and higher hospital expenses, according to multiple research. Antimicrobial/multidrug resistance development in *P. aeruginosa* can and does occur because horizontal gene transfer can and does lead to the acquisition of resistance genes, such as β-lactamases and aminoglycoside-modifying enzymes. However, it is more common for resistance to be due to chromosomal gene mutations (target site, efflux mutations).[17-18] Patients with weakened states, such as burns, are particularly vulnerable to infections caused by the Gram-negative opportunistic bacterium *Pseudomonas aeruginosa*, which is commonly seen in underdeveloped nations. Being naturally resistant to numerous medications and capable of developing resistance to all effective antibiotics, this bacterium is highly dangerous for people who become infected with it.[20] Many sections of the world are plagued with *Pseudomonas aeruginosa* and *Acinetobacter* spp. A prominent cause of infections acquired in intensive care units is *P. aeruginosa*. This bacterium has become more difficult to treat in a short period of time due to the discovery of almost every known antibiotic resistance mechanism in it. In many cases, these processes coexist, leading to XDR phenotypes and fewer possibilities for empirical treatment. This difficulty can be seen in the findings of a Chinese surveillance study: The study found that 7.5% of *P. aeruginosa* and 14.1% of *Acinetobacter baumannii* were PDR, meaning they could not be treated by an antibacterial agent. Due to the rarity of combined resistance data, the following information on *P. aeruginosa* concentrates on carbapenem resistance (induced by porin loss combined with enhanced b-lactamase expression and partially efflux). Carbapenem resistance varies widely across Europe, from 3% in the Netherlands to 50% or more in Romania and Greece. *P. aeruginosa* exhibits the same geographic gradient in resistance as other Gram-negative infections in Europe, with lesser resistance in the northwest and rising resistance in the southeast.[21] European data from 2008 show that 17% of *P. aeruginosa* isolates were resistant to three or more antipseudomonal antibiotic classes (piperacillin tazobactam, ceftazidime, fluoroquinolones, aminoglycosides, and carbapenems), with 6% demonstrating resistance to all five classes of antipseudomonal antibiotic tests.[11-22]

The low permeability of *P. aeruginosa* outer membrane (1/100 of the permeability of *E. coli's* outer membrane), the constitutive expression of various efflux pumps with broad substrate specificity, and the naturally occurring chromosomal AmpC β-lactamase make it intrinsically resistant to many structurally unrelated antimicrobial agents.[23] These β-lactams are known to cause natural resistance in the species, including penicillin G, aminopenicillins, and first and second generation cephalosporins. Due of the ease with which *P. aeruginosa* can pick up new resistance mechanisms, it might cause major therapeutic issues. Carboxypenicillins, ureidopenicillins, some third generation cephalosporins, all fourth-generation cephalosporin, monobactam aztreonam, and carbapenem imipenem and meropenem are among the drugs that can kill *P. aeruginosa*. There are a number of different types of basic resistance. A fourfold to eightfold increase in the MIC for the majority of β-lactams, including meropenem but not imipenem, characterizes this phenotype, which is sometimes referred to as "intrinsic resistance to carbenicillin".[24,25] There was no evidence of chromosomal AmpC β-lactamase synthesis above the fundamental level. Non-β-lactam antibiotic resistance such quinolones, trimethoprim, tetracycline, and chloramphenicol are all part of this phenotype. High MIC is due to low outer membrane permeability and activation or derepression of efflux mechanisms.[26] Only cephems (cefepime and cefpirome) and carbapenem resistance is influenced by the second phenotype. Derepression of the AmpC β-lactamase is responsible for the change's occurrence, and it is antibiotic-dependent.[25] During the third phenotypic phase of the infection, bacteria produce OXA-type β-lactamases and become increasingly resistant to penicillins (in particular ticarcillin, azlocillin,

and piperacillin). Resistance to carboxypenicillins and ureidopenicillins is determined by these narrow-spectrum oxacillinases, but not by resistance to cephalosporins, aztreonam, or moxalactam. The higher MICs to carbapenems characterize the fourth phenotype. Because strains with this phenotype have lower levels of OprD, a carbapenem-specific porin, their resistance to other β-lactams is unaffected.[25]

Resistant strains of *P. aeruginosa* can develop resistance to β-lactam antibiotics by increasing their enzyme synthesis. The amide link of the β-lactam ring is ruptured by penicilloyl-serine transferases (also known as β lactamases), hence the resulting compounds lack antibacterial action. The nucleotide and amino acid sequences of b-lactamases are used to classify them pharmacologically. Enzyme substrate and inhibitor profiles are used to classify enzymes into four functional categories (A–D). Metallo-β-lactamases (MBLs) require zinc for their action, whereas classes A, C, and D use a serine-based mechanism. ESBLs of classes A, B, and D are among the many b-lactamases discovered in *P. aeruginosa*, representing a large proportion of all four molecular classes.[25]

Antimicrobial resistance in *P. aeruginosa*: risk factors should be considered in the last few years, the prevalence of MDR has risen substantially, and it is now considered a serious global health problem. Several researches have looked into potential risk factors for the emergence of MDR strains. In a Brazilian case–control research, 142 patients with MBLs strains were compared to 26 patients with non-MBLs strains infected.[27,28] An ICU stay and a urinary tract infection were found to be major risk factors for MBLs infections using multivariate analysis. There was also a link between MBLs strains and an earlier onset of infection and a quicker course to death. A two-year retrospective research in Brazil, starting in 2010, looked at 54 patients with *P. aeruginosa* infections in the intensive care unit.[29] Three-hundred and seventy-seven percent of the patients tested positive for MDR *P. aeruginosa*, and twenty percent of the isolates tested positive for the blaSPM-1-like gene. MDR was found in patients who had spent an average of 87.1 days in the hospital. In China, a case–control surveillance research found that 54% of patients with *P. aeruginosa* infection had MDR *P. aeruginosa*. A tracheal intubation (OR: 2.21) and usage of carbapenems were also independent risk factors (OR 3.36). Hospitalization times and fatality rates increased when patients had MDR bacteria (49 versus 20%).[30] One study of 63 cases of carbapenem-resistant *P. aeruginosa* (CRPA) found that the APACHE II score at the time of CRPA infection, as well as CRPA's ability to form biofilm, might predict death in patients with CRPA bacteraemia independently of the other.[31,32] The APACHE II score was found to be an independent predictor of colonization in another investigation.[33]

Acinetobacter baumannii

Acinetobacter baumannii (*A. baumannii*) is a non-motile Gram-negative, aerobic, pleomorphic bacillus. *A. baumannii* is an opportunistic pathogen that commonly infects immunocompromised people, especially those who have spent an extended period of time in the hospital (more than 90 days). It is commonly found in aquatic habitats and can colonize the skin as well as be isolated in large numbers from the respiratory and oropharynx secretions of sick people. It is also commonly found in aquatic environments. It was previously categorized as a "red alert" human pathogen, causing concern in the medical community due to the wide range of antibiotic resistance it exhibits.[34] *A. baumannii* is a member of the genus *Acinetobacter*, is the most common cause of hospital-acquired infections in the world. Once considered a low-grade pathogen, this aerobic Gram-negative coccobacillus is now a successful pathogen that causes opportunistic skin, bloodstream, urinary tract, and other soft tissue infections which is a dramatic increase in the number of *A. baumannii* infections among Iraq and Afghanistan war veterans and military.[32-35] As an example of an infection caused by *A. baumannii*, inflammation of the airways in the lungs, also known as pneumonitis. An infection with *A. baumannii* can spread from the mouth or nose to the lungs. If a concern person is in the ICU or are on a ventilator, it could induce pneumonia. Blood infection: If the germ enters your vein through a catheter, it might cause a blood infection. Sometimes it happens when an illness from another part of your body spreads to your blood. Infection of the brain or spinal cord causes meningitis. Surgery to the brain or spine may cause this side effect. Another explanation is if you have a drain or shunt inside your brain. The kidneys, ureters, or bladder are infected with a urinary tract infection (UTI). The germ can enter your body through urination, and then spread throughout your body. Catheters meant to drain your urine can also be used to introduce the parasite. It can spread through any opening in the skin, including cuts and scrapes.[36] Multidrug-resistant (MDR) is a new pathogen in the hospital setting called *A. baumannii*, and it's been connected to a variety of infections including bacteremia, pneumonia, meningitis, and urinary tract infections. A common cause of infection outbreaks and an endemic pathogen connected with health care, the organism's capacity to thrive in diverse environmental conditions and stay on surfaces makes it a frequent source of infection.[37-39] The number of *Acinetobacter* species has grown to around 20 over the years. *A. baumannii* is one of the most prevalent *Acinetobacter* species seen in clinical settings, and it can cause a wide range of infections, such as respiratory tract infections, bacteremia, meningitis, and wound infections. The organisms that cause these infections have virulence factors such as porins on the outer membrane, capsules, lipopolysaccharides, phospholipase D, iron acquisition mechanisms, and regulatory proteins. *A. baumannii* is a drug-resistant bacterium, is known to produce life-threatening infections for which there are few effective treatments. Other medications still used to treat these infections, such as the carbapenem family of antibiotics are also losing their effectiveness. Because of an increase in carbapenem-resistant *Acinetobacter baumannii* (CRAB) found in previous years, treatment choices have become increasingly limited. Colistin, polymyxin B, and tigecycline are some of the few medications currently available to treat *Acinetobacter* spp. that are resistant to nearly all currently available antimicrobials.[36,40,41]

The worldwide prevalence of CRAB has recently increased, limiting treatment options and increasing morbidity and mortality rates.[42] Resistance to carbapenems in *A. baumannii* is caused by alterations in outer membrane proteins, efflux pumps, penicillin-binding proteins, and beta-lactamases. A, B, C, or D beta-lactamases. Class B (Metallo-b-lactamases, MBLs) and class D (OXA-type carbapenemases) carbapenemases produce carbapenem resistance.[43,44] OXA enzymes are encoded by blaOXA genes and are divided into eight subgroups: OXA-23, OXA-24, OXA-40, OXA-51, OXA-58, and OXA-143.[43,44] Intragenic sequences (IS) upstream of these genes alter their expression. These are essential for blaOXA

gene expression and carbapenem resistance.[34-43] Other MBL families, including VIM, IMP, GIM, SIM, and NDM enzymes, have been connected to the resistant phenotype in *A. baumannii*.[32-40] Finally, the significant prevalence of MDR *A. baumannii* in Saudi hospitals underscores the need of doing substantial research into the molecular basis of MDR and discovering novel therapeutics for CRAB.

Klebsiella pneumoniae

Klebsiella pneumoniae (*K. pneumoniae*) is a non-motile gram-negative, encapsulated, and environmental bacterium that has been correlated to pneumonia in patients with alcohol use disorder or diabetes mellitus. Oropharynx and gastrointestinal (GI) tract mucosal surfaces are typical sites of colonization for the bacteria. Carl Friedlander published the first description of *Klebsiella pneumoniae* in the journal in 1882, and the rest is history.[45] After removing the bacterium from the lungs of people who had died of pneumonia, he classified it as an encapsulated bacillus. *Klebsiella* was originally known as Friedlander's bacillus and was not given the name *Klebsiella* until 1886.[46] Today, *K. pneumoniae* is the leading cause of hospital-acquired pneumonia in the United States, with a prevalence of 3–8% of all nosocomial bacterial infections being caused by the organism.[47,48] MDR Enterobacteriaceae healthcare-associated infection is extremely diverse. As an example, *Escherichia coli* and *Klebsiella pneumoniae* both provide significant therapeutic challenges because of their fast resistance development as well as their high incidence of multidrug resistance.[49] Despite the fact that *K. pneumoniae* has greater MDR rates than *E. coli* and carbapenem resistance has lately increased rapidly in a number of places.[50] Contemporary public health faces a major threat from the proliferation of antibiotic resistance determinants and multidrug-resistant microorganisms. The bla_{NDM-1} determinant from the Indian subcontinent has spread rapidly since its discovery in early 2008 from a carbapenem-resistant *Klebsiella pneumoniae* urine isolate, resulting in its prevalence across a wide range of bacterial species all over the world. During the months of January to June of 2010, the first reports of NDM-1-producing bacteria were made in the USA. One of the first three bla_{NDM-1}-containing strains found in the United States was also a *K. pneumoniae* urine isolate from a patient who had just received medical care in India, similar to the initial bla_{NDM-1}-containing strain. The only other strain in the American Type Culture Collection repository has bla_{NDM-1}, and this one does (ATCC BAA-2146). For efficient infection control and informed therapy options, research and public health efforts including early and rapid detection of bla_{NDM-1}-containing strains must be coordinated. This has led to major implications for the dissemination of this carbapenemase.[51] Infections caused by carbapenem-resistant *Klebsiella pneumoniae* (CRKp) are still a major cause of morbidity and mortality around the world. Against CRKp, only a few antimicrobials are still active. Aminoglycosides, tigecycline, and the recently approved ceftazidime/avibactam antibiotics are examples of this class of medication. Polymyxins such as colistin, as well as other antibiotics, are effective therapeutic options. Colistin is frequently used with other antibiotics, such as tigecycline, meropenem, gentamicin, or fosfomycin, in cases of severe CRKp infections. Colistin-resistant *Klebsiella pneumoniae* carbapenemase (KPC)-producing bacteria are being found over the world as the drug's use increases.[52,53] ColR has emerged in CRKp, and it presents clinicians and patients with an additional treatment obstacle that could send them back to a time before antibiotics were widely used. There was a comparison made between the outcomes of patients with ColR CRKp and those with ColS CRKp using data from the multicenter Consortium on Resistance against Carbapenems in *Klebsiella pneumoniae* (CRACKLE).[52]

Conclusions and Recommendations

Risk for hospital-acquired illnesses is based on infection control methods at the institution, the patient's immunological condition, and the presence of certain pathogens in the local population. Immunosuppression, older age, duration of hospital stay, numerous underlying comorbidities, mechanical ventilation, recent invasive surgeries, indwelling devices, and intensive care unit stay were identified as risk factors (ICU). During the 1990s, hospitals have taken hospital-acquired pollutions seriously. Some hospitals have implemented infection monitoring and surveillance systems, as well as comprehensive infection avoidance tactics, in an effort to reduce the incidence of hospital-acquired infections. The effect of hospital-acquired infections is felt not only at the patient level, but also at the community level, since they are connected to multidrug-resistant pollutants. Identifying patients with risk factors for hospital-acquired infections and multidrug-resistant pathogens is crucial for preventing and minimizing these illnesses. All persons and organizations providing health care are responsible for preventing nosocomial infections. All parties must collaborate to limit the risk of infection for patients and workers. This consists of individuals providing direct patient care, management, supply of supplies and goods, and training of health professionals. Effective infection control programs include surveillance, prevention, and staff education. Also, national and regional levels of assistance must be successful. High prevalence of MDR and XDR strains is a serious concern in hospital wards. This review highlights the need for monitoring and strict antimicrobial stewardship policies and strong microbiological surveillance procedures in the hospital.

Conflict of Interest

None.

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