Chapter

Hospital-Acquired Pneumonia

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Abstract

Pneumonia acquired during hospitalization is called nosocomial pneumonia (NP). Nosocomial pneumonia is divided into two types. Hospital-acquired pneumonia (HAP) refers to hospital-acquired pneumonia, whereas ventilator-associated pneumonia (VAP) refers to ventilator-associated pneumonia. Most clinical literature stresses VAP's importance and associated mortality and morbidity, whereas HAP is not given enough attention even while being the most common cause of NP. HAP, like VAP, carries a high mortality and morbidity. HAP is the commonest cause of mortality from hospital-acquired infections. HAP is a common determinant for intensive care unit (ICU) admits with respiratory failure. Recent research has identified definite risk factors responsible for HAP. If these are prevented or modified, the HAP incidence can be significantly decreased with improved clinical outcomes and lesser utilization of the health care resources. The prevention approach will need multiple strategies to address the issues. Precise epidemiological data on HAP is deficient due to limitations of the commonly used diagnostic measures. The diagnostic modalities available in HAP are less invasive than VAP. Recent infectious disease society guidelines have stressed the importance of HAP by removing healthcare-associated pneumonia as a diagnosis. Specific differences exist between HAP and VAP, which are gleaned over in this chapter.

Keywords: hospital-acquired pneumonia (HAP), ventilator-associated pneumonia (VAP), ICU, prevention

1. Introduction

Nosocomial pneumonia (NP) that occurs during a patient's hospital course has been subclassified into hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP). As per the latest Infectious Diseases Society of America (IDSA) and American Thoracic Society guidelines (ATS) [1], the category healthcare-associated pneumonia (HCAP) has been abandoned. The term NP and HAP should not be used interchangingly as before. HAP should be used only for pneumonia that occurs >48 h after admission to a hospital. VAP refers to pneumonia occurring >48 h post-intubation [2]. HAP is the most frequent hospital-acquired infection (HAI) [3]. As per the latest study done in the United States of America (USA), HAP prevalence in ICU was more frequent than VAP, and more than 75% of these patients developed severe respiratory failure due to pneumonia resulting in intubation and mechanical ventilatory support [4]. It is unknown whether the above trend is similar across all medical centers in the USA or is observed only in a few medical centers. Tertiary

medical centers may have a different prevalence rate than other medical centers due to the higher presence of immunosuppressed patients (post-transplant). The lack of effective HAP surveillance systems in the USA and other countries adds to this tenuous issue. Also, the lack of definitive diagnostic criteria makes it difficult to identify HAP patients on the floor and in intensive care units, as fever and cough can have multiple diagnostic possibilities postadmission to a hospital.

2. Epidemiology

HAP can occur in both patients with or without risk factors, and it is critical to realize that all acute care patients have an increased risk of HAP [5]. Specific patient subsets carry an increased risk than others, including elderly patients, chronic lung, cardiac and renal disease, hepatic cirrhosis, obesity, diabetes mellitus, cancer, neurological conditions such as stroke and dementia, malnutrition, and immunosuppressed patients [6, 7]. Specific therapeutic intervention modalities, including medications and procedures such as intubation, gastric tube placements, can increase the risk of HAP. Clinical literature on HAP inside the ICU is suboptimal, whereas on HAP outside the ICU is minuscule. NP accounts for around 21 admits per 1000 admissions to a hospital [8]. NP is responsible for close to 22% of HAI in the USA, and about 61% are HAP compared to VAP [9]. NP results in significant clinical outcomes such as increased healthcare costs, extended hospital stay, excess utilization of health care resources, and higher mortality and morbidity [10]. The actual prevalence rates of HAP and VAP are unknown; however, recent studies allude to a greater prevalence of HAP than VAP by a ratio of close to 2:1 in favor of HAP [11, 12]. A recent state study from Pennsylvania revealed that HAP risk factors and resulting complications are identical to those seen in VAP but were associated with an unfavorable higher economic cost and similar mortality [11]. Recent studies indicate an approximate incidence of 1.22 to 8.9 per 1000 patient days [5, 6, 9, 13, 14]. The total acute care cost for HAP is close to 40,000 dollars, with a hospital stay of 4 to 15.9 days, and the HAP influence on mortality was more significant than VAP [11, 13, 14]. Also, HAP patients, due to their increased occurrence, had a net increased economic cost than VAP and a higher need for postdischarge care [11, 14]. However, this cost did not include the interinstitutional transfer costs involved [14].

3. Etiology and risk factors

As patients diagnosed with HAP are not intubated, they face multiple challenges, including an inability to perform minimally invasive procedures to obtain microbiological specimens from the lower airway leading to the absence of microbiological data and ineffective initial antimicrobial treatment. In a large European trial involving 27 ICU units among HAP patients, only 54.8% of patients had positive microbiology data. *Enterobacteriaceae* are the most frequent cause, followed by *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* [15]. In another study, the microbial causes were similar between HAP and VAP except for an increased occurrence of *Streptococcus pneumoniae* in HAP patients [16]. 80% of cases were caused by *Klebsiella spp., Enterobacter spp.*, *Escherichia coli*, *Staphylococcus aureus*, *Acinetobacter spp.*, and *Pseudomonas aeruginosa* per the clinical data registered in the antimicrobial surveillance program SENTRY [17]. Also, in this study, severe sepsis and pneumonia

occurred only in centers with >25% Multi-drug Resistance (MDR) prevalence, even in those lacking risk elements and early pneumonia. Upon reviewing the data mentioned above, gram-negative bacilli (GNB) cause most of these infections and are frequently resistant to antibiotics, making an empirical antibiotic decision difficult. In transplant patients, the microbial etiology differs based on the transplant type, duration post-transplant, and the antirejection mediations they are currently on. In hematopoietic stem cell transplants, bacterial causes were the highest, followed by fungal and viral [18]. Among the bacterial causes, the most common cause was *Escherichia coli*, *Pseudomonas aeruginosa*, and *Streptococcus pneumoniae*. GNB was the most frequent in solid organ transplants, especially *Pseudomonas aeruginosa*, *Enterobacteriaceae*, followed by

Invasive MSSA[*] infection risk factors	**MRSA[**] HAP risk factors**
1. Cardiac disease	1. Tobacco abuse
2. Diabetes mellitus	2. Illicit drug abuse
3. Cancer	3. Recent hospitalization <90 days
4. Chronic obstructive pulmonary disease	4. Recent antibiotics
5. Hemodialysis	5. Chronic obstructive pulmonary disease
6. Stroke	6. Liver disease
7. Intravenous drug abuse	7. HIV infection
8. Rheumatoid arthritis	
9. Human immunodeficiency viral infection	
10. Peritoneal dialysis	
11. Solid organ transplantation	
12. Systemic lupus erythematosus	

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[*]*MSSA—Methicillin-sensitive* Staphylococcus aureus.
[**]*MRSA—Methicillin-resistant* Staphylococcus aureus.

Table 1.
Invasive MSSA infection risk factors, MRSA HAP risk factors.

1. Prior infection with *pseudomonas spp*.
2. *Pseudomonas spp*. colonization
3. Very severe COPD
4. Bronchiectasis
5. Tracheostomy
6. Neutropenia
7. Burns
8. Cystic fibrosis
9. Long term acute care residents
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Table 2.
Pseudomonas spp. *HAP risk factors.*

Staphylococcus aureus, often with an MDR profile [19]. The microorganisms responsible vary based on the patient population, MDR risk factors, geographical location, and duration of hospital stay before disease onset [2]. An essential factor to recognize is identifying any Multi-drug resistance organism (MDRO) risk factors, clinical severity, and local ecology before empirical antibiotic therapy. **Tables 1–3** reveals the risk factors for *Staphylococcus aureus* [20, 21], *Pseudomonas aeruginosa* [22], and *Acinetobacter baumannii* [23–26].

A prospective study has revealed the intrinsic and extrinsic risk factors for HAP in non-ICU patients, as shown in **Table 4** [6]. Demographically age > 60 years and males are at higher risk of acquiring HAP.

1. Long term acute care residents
2. Prior colonization/infection
3. Longer hospital duration stay
4. Prior antibiotics use
5. Acinetobacter skin infections
6. Poor healthcare worker hygeine
7. Contamined procedure equipment
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Table 3.
Acinetobacter spp. *HAP risk factors.*

Intrinsic risk factors	**Extrinsic risk factors**
1. Cancer	1. Duration of hospitalization >5 days
2. Chronic obstructive pulmonary disease	2. Prior antibiotic therapy
3. Diabetes mellitus	3. H2 antagonist
4. Congestive heart failure	4. Steroids
5. Chronic renal failure	5. Antacids
6. Depression	6. Chemotherapy
7. Neutropenia	7. Prior endotracheal intubation
8. Obesity	8. Nasogstric tube
9. Malnutrition	9. Nebulization
10. Liver cirrhosis	10. Abdominal surgery
11. Human immunodeficiency virus infection	11. Prior ICU admission
	12. Thoracic surgery
	13. Head and neck surgery
	14. Tracheotomy
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Table 4.
Intrinsic and extrinsic risk factors for HAP in non ICU patients.

4. Pathophysiology

The upper airway and the oropharynx are usually colonized with nonpathogenic microorganisms, including the virulent *Staphylococcus aureus* and *Streptococcus pneumoniae*, and anaerobes. The lower airway microbiome is not entirely void of bacteria, as thought before [27]. The lower airway microbiome changes during chronic lung disease or prolonged immunosuppression. Within a few days post-admission, the upper airway and the oropharynx flora changes on exposure to the hospital ecology and get colonized with MRSA (*Methicillin-sensitive Staphylococcus aureus*) and GNB [28, 29]. Most HAP occurs after aspiration of the oropharyngeal flora except for few bacterial microorganisms, viral and fungal microorganisms, which occur via respiratory droplets or inhalation. Once inhaled or aspirated, the intact mucociliary clearance, mucociliary and alveolar defense will try to clear it up [30–33]. They are often successful, but in cases with a large aspiration in a healthy patient or

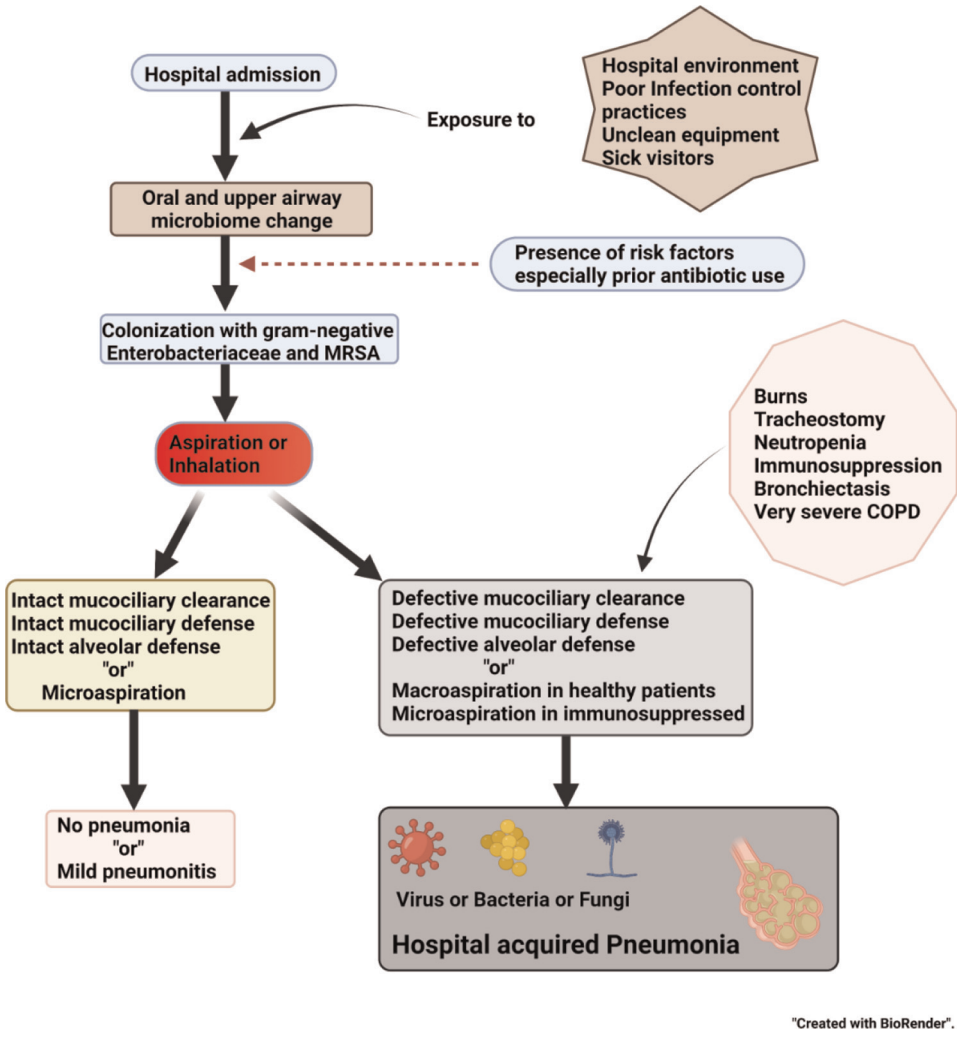

Figure 1.
Pathophysiology of HAP.

Clinical symptoms	Clinical signs
1. Fever	1. Tachycardia
2. Dyspnea	2. Hypotension
3. Cough	3. Tachypnea
4. Tachypnea	4. Hypoxia
5. Chest pain	5. Rales
6. Purulent sputum	6. Wheezing
7. Hypothermia	7. Use of accessory respiratory muscles
8. Generalized weakness	8. Absent/decreased breath sounds
9. Confusion	9. Altered mental status
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Table 5.
Clinical features of hospital-acquired pneumonia.

microaspiration in an immunosuppressed individual, the protective mechanisms are overwhelmed and result in HAP with significant inflammation and systemic signs. This entire process has been outlined in **Figure 1**.

5. Clinical features

The clinical features of HAP have been summarized as follows in **Table 5**.

6. Diagnosis and differential diagnosis

Due to the lack of diagnostic criteria, clinical features need to be supplemented by imaging or laboratory tests for a HAP diagnosis. Imaging is often a portable or a two-view chest radiograph that reveals new pulmonary infiltrates, cavitation, abscess, or pleural effusion. Chest computed tomography (CT) is a gold standard in comparison and has better sensitivity than chest X-rays [34]. Recently, bedside ultrasound has been used to identify new pulmonary infiltrates with 94% sensitivity and 96% specificity [35]. A retrospective trial has revealed that biomarkers procalcitonin and C-reactive protein correlate well with HAP severity and could be a better prognostic marker for mortality and morbidity than neutrophil/lymphocyte count ratio [36]. A complete blood count may demonstrate leukocytosis. The differential count is essential in identifying any neutrophilia, neutropenia, eosinophilia, and a peripheral smear may demonstrate Dohle bodies that are more suggestive of ongoing infection. Microbiological workup can be invasive or noninvasive. Blood cultures with the help of MALDI BioTyper and FilmArray BCID can help rapidly identify the bacteria [37]. In transplant patients, a fungal blood culture would be ideal. Urine legionella and Streptococcal antigens can help identify the cause of pneumonia. Serum Aspergillus antigen assay and β-D-glucan assay is a must in transplant and immunosuppressed individuals when suspected. Nasopharyngeal swab polymerase chain reaction (PCR), also called a respiratory pathogen panel, can be utilized to identify some of the common

respiratory bacterial and viral pathogens causing community-acquired pneumonia, which can also cause HAP due to significant exposure prior to admission and in the hospital.

The sputum gram stain, sputum specimen PCR, and culture should be done to identify the suspected etiological agent. If there is a lack of sputum, production then it can be induced by inhaled hypertonic saline. Sputum PCR using BioFire FilmArray Pneumonia or Pneumonia plus panel yields excellent sensitivity and specificity but must be adopted judiciously, and it could provide appropriate clinical information for antimicrobial stewardship [38, 39]. It can also detect atypical bacteria, common viral causes of pneumonia, common mechanisms of resistance and provide semiquantitative results for the common colonizers [40]. It provides valuable data for the clinician to deescalate the antibiotics to a narrow spectrum. This PCR test does not detect oral anaerobes, and they need to be considered with positive imaging and a negative PCR test to cover them with appropriate antibiotics. The PCR test should be done early in the clinical course to avoid false negatives and must be corroborated with the culture as much as possible. A sputum fungal culture or stain also might be helpful in transplant patients.

The invasive strategy involves performing a fibreoptic bronchoscopy, obtaining a bronchioalveolar lavage (BAL) sample, and performing BAL tests, including gram stain, fungal stain, cytology with methenamine stain, and quantitative culture (bacterial and fungal). BAL Aspergillus antigen assay, β-D-glucan assay, fungal and viral PCR assays can detect the causative agent in immunosuppressed or transplant patients. Invasive tests are done seldomly in stable patients as most of these patients are sick, unstable and the procedure may clinically deteriorate them [41]. If the patient during his clinical course gets intubated, then a BAL should be obtained to obtain more clinical information.

1. Two or more serial chest imaging test results with at least one of the following new and persistent or Progressive and persistent (Radiological criteria)
*Infiltrate/Consolidation/Cavitation
PLUS
2. Atleast one of the following (Systemic criteria)
• Fever (>38.0°C or > 100.4°F)
• Leukopenia (≤4000 WBC/mm^3) or leukocytosis (≥12,000 WBC/mm^3)
• For adults ≥70 years old, altered mental status with no other recognized cause
PLUS
3. And at least two of the following (Pulmonary criteria)
• New onset of purulent sputum or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements
• New onset or worsening cough, or dyspnea, or tachypnea
• Rales6 or bronchial breath sounds
• Worsening gas exchange (for example: O_2 desaturations (for example: $PaO_2/FiO_2 \leq 240$)7, increased oxygen requirements, or increased ventilator demand)

Table 6.
National health safety network definition of pneumonia (NHSN PNEU).

1. Pulmonary contusion
2. Pulmonary inhalation injuries
3. Atelectasis
4. Pleural effusion
5. Pulmonary edema
6. Pulmonary hemorrhage
7. Drug-induced pneumonitis
8. Pulomary infarct/embolism
9. Vasculitis
10. Primary or secondary pulmonary neoplasm

Table 7.
Differential diagnosis of HAP.

The betaLACTA test (BLT) detects GNB insensitivity to third-generation cephalosporins due to carbapenemases, ESBL (extended-spectrum beta-lactamases), and beta-lactamases from acquired AmpC carbapenemases in less than 20 min after exposure to respiratory bacterial cell pellets via chromogenic analysis [42]. The test detects GNB resistance via a colorimetric indicator and can quickly be used for antibiotic de-escalation [43]. It is currently being evaluated for its clinical efficaciousness in France's multicenter randomized controlled trial (RCT) called BLUE-CarbA [44].

A clinical diagnosis of HAP is currently considered with a new lung infiltrate and two of the four findings, including new-onset temperature > 38 degrees celsius, purulent sputum, and leukocytosis or leukopenia [45]. Most clinical diagnostic scores, including modified clinical pulmonary infection score (CPIS), the older National safety health network (NHSN) pneumonia definition, and the new infection-related Ventilator-associated complication (IVAC), have been used extensively in VAP and not in HAP. NHSN does suggest using the pneumonia definition for nonventilated adult patients for surveillance purposes (**Table 6**). However, the long-term clinical utility of its use is unknown due to its lack of accuracy and consistency in VAP [46].

Clinical conditions that may simulate HAP and may need to be considered part of the differential diagnosis are mentioned in **Table 7**.

7. Treatment

Initial inappropriate antibiotic regimens and MDRO are independent indicators of ICU mortality and related to a longer mechanical ventilation duration [47]. Physicians always face a clinical scenario where they have to treat a patient with no lower respiratory specimen with the possibility of pending acute respiratory failure requiring mechanical ventilation [41]. Empirical antibiotic therapy can be based either on institutional epidemiology or a surveillance culture report updated annually. Although they yield similar results, the use of surveillance culture report results in reduced broad-spectrum antibiotics uses even in the presence of higher MDRO risk factors [48]. Individual patient risk factors need to be considered before an initial empirical regimen is started for HAP [49]. A suggestion is to use the local antibiogram in deciding the initial regimen. Most regimens include a broad-spectrum gram-positive

coverage (vancomycin or linezolid) and a gram-negative coverage (carbapenem or fourth-generation cephalosporin or a piperacillin-tazobactam). It is prudent to use an antipseudomonal agent to cover gram-negative bacteria in the empirical regimen. MRSA screening of nares has a 96.1% negative predictive value for respiratory cultures [50]. Gram-positive bacterial coverage can be deescalated to MSSA coverage with a negative MRSA nasal screen if the clinical condition warrants it.

For de-escalation, at 48 to 72 h postadmission, procalcitonin plus C-reactive protein and a positive microbiological workup assist the clinical criteria [2]. De-escalation involves a transition from broad-spectrum to narrow-spectrum antimicrobial therapy. For atypical organism coverage, if suspected, rarely responsible for HAP, azithromycin or fluoroquinolone, or doxycycline can be used in addition to the empirical therapy. For *P. aeruginosa* HAP with no susceptibility results or absence of septic shock or high death risk, dual antipseudomonal coverage is indicated [2]. If the susceptibility pattern has resulted and in the absence of septic shock and increased risk of death, monotherapy is appropriate. *P. aeruginosa* HAP with carbapenemase resistance (CRE) can still be treated with ceftolozane/tazobactam combination as its primary resistance is via porin channels [51]. With ESBL GNB causing HAP, the recommended therapy is carbapenems with suggested alternatives, including ceftolozane/tazobactam combination. With *Acinetobacter spp.*, the treatment is based on antimicrobial susceptibility and usually involves more than one drug. CRE GNB is treated with Ceftazidime/avibactam, and Aztreonam is added to combination in GNB

Active measures taken	**Effectiveness of measure**
A. Exposure reduction: All the below-mentioned measures need further evaluation in HAP patients [54].	
Limit admission to hospital as much as possible	Increased hospitalization duration is associated with increased sepsis warning scores and increased exposure to HAP pathogens [55]. The risk in elderly patients increases at a rate of 0.3% per day [56]. Decreased duration of hospitalization results in decreased exposure and risk; however, this needs prospective assessment.
Healthcare worker and equipment hygiene	It prevents microbial spread between patients, health care workers, and essential equipment and improves VAP and catheter-associated bloodstream infection rates [57]. Stethoscopes and portable procedure equipment cleaning with chlorhexidine or alcohol-based sanitizer are ideal [58, 59]. The use of a portable stethoscope separately for each patient is another option [60]. Minimally invasive procedure equipment such as endoscopes and bronchoscopes should be sterilized with stringent protocols. Low compliance is frequent in healthcare workers and needs to be improved with structured educational programs and timely reinforcements [61].
Isolation measures	Standard isolation precautions such as universal gowns and gloves are ineffective in preventing the transmission of infections caused by MDRO [62]. However, they are highly effective in preventing *Clostridium difficile (C. difficile)* transmission [63]. Droplet precautions in hospitalized influenza infections prevent its spread.
B. Aspiration reduction: As mentioned above, the below-mentioned measures need validation in HAP patients.	
Prevent and reduce xerostomia	Xerostomia or oral dryness correlates with fever in dysphagia patients, but its association with HAP is unknown [64]. Also, the effect of xerostomia prevention and treatment with sialogogues on HAP incidence and prevalence is unknown [65].

Active measures taken	Effectiveness of measure
Timely identification of dysphagia	Identifying patients with a higher risk of dysphagia promptly by a higher screening adherence results in lower HAP rates [66]. This is especially important in patients with neurological disorders. Dysphagia evaluation by a speech therapist can lead to modified diets in specific population subsets with a lower incidence of pneumonia [67].
Feeding via enteral tubes	Jejunostomy tubes compared to gastric ones result in lower VAP and HAP rates [68]. The use of a motility agent has lead to variable results in a systematic review, and its benefit is questionable [69].
Patient position modification	A semi-recumbent position (30° to 45°) during feeding decreases acid reflux and the risk of aspiration with a decline in VAP rates [70].
Mobilization	Earlier mobilization stops the functional decline, improves airway clearance, and prevents HAP [71]. Family member's training helps in extending this benefit outside of the healthcare environment [72].
C. Active interventions	
Oral hygiene	Bad oral hygiene results in increased colonization with airway pathogens and periodontal disease [73]. It can diminish cough reflex and impair airway hygiene leading to pneumonia [74, 75]. Interventions to improve oral hygiene are the best known cost-effective preventive strategy for HAP [5, 76]. Adequate training of nursing staff in oral care practices is critical with timely reinforcements.
Decontamination of oral, digestive, and skin	Skin decontamination with chlorhexidine decreases VAP, HAI but its effect on HAP is unknown [77]. Oral decontamination with chlorhexidine diminishes VAP rates and increases mortality; however, its implication on HAP is unknown [12, 78]. Selective digestive decontamination (SDD) with oral, topical, and intravenous antibiotics decreased VAP and is thought to be adequate in HAP [79]. SDD use was in countries with lower antibiotic resistance levels, and its long-term effects are unknown [12].
Vaccination	Vaccination against hospital pathogens is ineffective [80], whereas monoclonal antibodies have shown promise adjunctively with antibiotics in early trials for *Staphylococcus aureus* and *Pseudomonas aeruginosa* [81].
Medications and other factors	As medications preventing gastric-acid secretions are linked to increased HAP rates, preventing their indiscriminate use is necessary [82]. Adequate glucose control preventing hypo and hyperglycemia is critical in halting airway colonization and pneumonia risk [83, 84]. Probiotics may decrease the HAP rate; however, they have not been evaluated in HAP.
Airway hygiene	When done preemptively in postoperative and hospitalized pneumonia patients, chest physical therapy has revealed modest preventive effects [85, 86].
Respiratory support	Noninvasive ventilation (NIV) decreased nosocomial pneumonia and improved outcomes in specific patient subsets [87]. Although it allows for better airway clearance and comfort, high-flow nasal cannula use did not decrease HAP incidence in two small randomized controlled trials [88, 89]. Recent helmet use in NIV did not decrease HAP rates compared to facemask [90].
Staffing practices	Increased nursing staff to patient ratio results in lower HAP and HAI rates [91]. The presence of daytime intensivists correlates with improved mortality overall [92]. The effect of 24 h physician staffing on the HAP rates is unknown.

Table 8.
HAP preventive measures.

carrying Metallo-carbapenemase. The usual duration of treatment is around 7 days as in VAP with some exceptions, which include MSSA, MRSA, nonfermenting GNB such as *Pseudomonas*, *Stenotrophomonas*, *Acinetobacter*, and *Burkholderia spp*., which have a higher rate of recurrence with 7 days of therapy (this data extrapolated from VAP studies) [52]. Regarding MSSA and MRSA HAP, the duration mentioned above is a recommended expert opinion due to the lack of RCTs on the course of therapy (7 vs. 14 days) [53]. Other exceptions to the seven-day course could be patients with immunosuppression and necrotizing pneumonia. Antimicrobial treatment should be based on the pharmacokinetics and pharmacodynamic data of the individual antimicrobial to avoid unwanted side effects.

8. Prevention

The utilization of any preventive measures to halt HAP should effectively alter the pathophysiology of the disease. Multiple measures have been carried out over the last few decades to prevent HAP or, preferably, VAP with variable degrees of success (**Table 8**).

A constant surveillance system absence regarding HAP has prevented effective detection and monitoring of HAP rates in the USA. An objective assessment is hampered by the lack of standard diagnostic criteria, microbiologic and diagnostic coding data [54]. Also, only a few preventive measures have been validated, and the remaining lack adequate clinical data for physicians to implement them successfully. It requires multidisciplinary team involvement for the effective implementation of these preventive measures.

9. Conclusion

The administratively coded data (ACD) used for billing is limited, and its accuracy is imprecise in HAP detection and surveillance [14]. A better approach to this problem will be to use proven assessed techniques, and this practice should be utilized in HAP detection. The approach should start with creating a specific diagnostic criterion followed by evidence-based guidelines to help in decreasing its incidence and prevalence with additional stress on earlier detection and prevention.

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Acronyms and abbreviations

NP	Nosocomial pneumonia
HAP	Hospital-acquired pneumonia
VAP	Ventilator-associated pneumonia
ICU	Intensive care unit
IDSA	Infectious Diseases Society of America
HCAP	Healthcare-associated pneumonia
HAI	Hospital-acquired infections
USA	United States of America
MDR	Multi-drug Resistance
MDRO	Multi-drug Resistance Organism
HIV	Human Immunodeficiency virus
MSSA	Methicillin-sensitive *Staphylococcus aureus*
MRSA	Methicillin-resistant *Staphylococcus aureus*
COPD	Chronic obstructive pulmonary disease
GNB	Gram-negative bacilli
CT	Computed tomography
MALDI	Matrix-assisted laser desorption ionization
BCID	Blood Culture ID Panel
PCR	Polymerase chain reaction
BAL	Bronchioalveolar lavage
BLT	betaLACTA test
ESBL	Extended-spectrum beta-lactamase
RCT	Randomized controlled trial
CPIS	Clinical pulmonary infection score
NHSN	National health safety network
IVAC	Infection-related Ventilator-associated complication
CRE	Carbapenemase resistance
SDD	Selective digestive decontamination
NIV	Noninvasive ventilation
ACD	Administratively coded data

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