Health promotion, risk stratification, and treatment options to decrease hospitalization rates for community-acquired pneumonia in adults

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Abstract

Purpose: Community-acquired pneumonia (CAP) is a serious illness and hospitalization for this illness is expensive. There is much the nurse practitioner (NP) can do to prevent and manage this illness.

Data sources: Review of current literature, medical/nursing references, and data from the healthcare utilization project (HCUP).

Conclusions: The use of health promotion, risk stratification, and current evidence-based treatment guidelines can help to decrease hospitalization rates for CAP for adults.

Implications for practice: NPs are experts at health promotion and evidence-based practice. Adhering to these practices and using risk stratification, NPs can help to further decrease hospitalization rates for CAP lowering healthcare costs related to this serious illness.

Introduction

Pneumonia is a serious illness with life-threatening implications and is a leading cause of morbidity and mortality. As of 2010, influenza and pneumonia are the ninth cause of mortality in the United States (Murphy, Xu, & Kochanak, 2012). There are approximately 5.6 million cases of community-acquired pneumonia (CAP) annually with an average of 20% of those patients requiring hospitalization. CAP is the leading cause of death because of an infectious disease with 2%–21% mortality and an increase to 50% mortality for patients hospitalized with severe disease (Genne’ et al., 2006).

Many patients are hospitalized for the treatment of CAP because it is such a deadly disease. Health promotion strategies, risk stratification, and evidence-based treatment options are useful in lowering rates of hospital admission related to CAP. Nurse practitioners (NPs) on the frontlines in primary care can make a critical difference in the implementation of health promotion strategies aimed at decreasing overall pneumonia rates. Furthermore, NPs can use risk stratification tools to decide who is and is not a good candidate for outpatient therapy with CAP.

Epidemiology

According to the National Center for Health Statistics (2012), hospital discharges for pneumonia in the United States are approximately 1.1 million annually with an average hospital stay of 5.6 days. The annual number of deaths resulting from pneumonia is 55,774 or 16.5 deaths per 100,000 of the U.S. population. Pneumonia accounts for 3.4% of inpatient deaths (National Center for Health Statistics, 2012).

The main causes of death related to CAP are because of pneumonia-related complications, such as sepsis, multi-organ failure, refractory hypoxemia, and shock (Restrepo & Anzueto, 2009).

The estimated cost of treating CAP per year in the United States is approximately $12.2 billion dollars (Asche, McAdam-Marx, Seal, Crookston, & Mullins, 2008). Inpatient treatment is approximately 20 times as much as outpatient care (Labarere et al., 2007). Hospitalizations because of CAP have an average cost of care from $7500 to $10,227 per incident (Lutfiyya, Henry, Chang, & Reyburn, 2006; Restrepo & Anzueto, 2009). The cost for outpatient treatment is much less and may be as little as $150–$350 per patient (Lutfiyya et al., 2006).
Risk factors

Individuals at risk for CAP include those who smoke, have comorbid conditions, and antibiotic treatment or hospitalization within the past 3 months. Comorbid conditions include asthma, lung cancer, chronic obstructive pulmonary disease, diabetes, alcoholism, chronic renal disease, liver failure, heart failure, malnutrition, human immune deficiency virus, and chronic steroid use (Centers for Disease Control and Prevention [CDC], 2012; Mandell et al., 2007).

According to Mandell et al. (2007) the elderly are more at risk for CAP because of increased number of comorbidities, decreased mucociliary clearance, diminished cough reflex, increased incidence of aspiration, increased colonization with gram-negative organisms, and depressed immune systems. Alcohol increases CAP risk because of cough suppression, increased colonization of gram-negative organisms, and immune suppression. Cigarette smoking, long known as a risk factor for CAP, impairs mucociliary action and macrophage activity (Mandell et al., 2007).

Pathogenesis

Pneumonia is an infection of the lungs by organisms, such as bacteria, fungi, and viruses. This typically starts with colonization of the offending organism in the nasopharyngeal area, but can also occur when infectious droplets are directly inhaled and as a secondary infection because of sepsis or bacteremia. If the host has altered protective mechanisms, such as impaired ciliary action, over production of mucous, immobility or other immune deficiencies, they have higher risk of developing CAP. When organisms enter the alveoli in the lung, an immune response is triggered (Brashers, 2008; Chesnutt & Prendergast, 2011; Ranganathan & Sonnappa, 2009).

The type and virulence of the offending organism, in part, determines the extent of the immune response, which can be mild to severe. Organisms with low virulence are typically engulfed by macrophages setting off only a mild immune response. If the offending organism is great in numbers or is highly virulent, a series of immune responses occur including the release of inflammatory mediators, cellular infiltration, and immune activation. The more involved the immune response the more symptomatic the patient becomes (Brashers, 2008; Chesnutt & Prendergast, 2011; Ranganathan & Sonnappa, 2009).

Viruses can cause primary pneumonia or predispose an infected individual to secondary bacterial pneumonia. The presence of viruses not only initiates an immune response, but also damages the host by destroying ciliated epithelial cells, goblet cells, and bronchial epithelium. This naturally weakens host defenses as bronchial epithelium sloughs in the presence of diminished mucociliary clearance. Decreased clearance of pulmonary secretions creates the perfect breeding ground for host bacteria leading to a secondary bacterial infection (Brashers, 2008).

Patients become symptomatic from the body’s response to invading organisms and from the destruction of cells from viral organisms and/or through the release of toxins from bacterial cell walls. As white blood cells and other chemicals from the immune system arrive at the site of the invading organism they fill the alveoli in the lungs. This impairs oxygenation and produces the symptoms typical of pneumonia, cough, hypoxia, fever, chills, tachypnea, fatigue, and malaise (Brashers, 2008; Chesnutt & Prendergast, 2011; Ranganathan & Sonnappa, 2009).

CAP is one of five classifications for pneumonia and is defined as an acute infection of the pulmonary parenchyma acquired in the community setting. To meet the criteria for CAP a patient must not have been hospitalized within 14 days or onset of symptoms must be within 4 days of hospitalization, and must not be a resident in a long-term care facility (Nazarian, Eddy, Lukens, Weingart, & Decker 2009).

In many cases of pneumonia, the causative organism is not identified because of the success of empirical treatment and risk stratification. Testing for the causative organism is typically reserved for the patient with a higher acuity of illness from CAP (Restrepo & Anzueto, 2009). Enough research has been carried our to date to identify the most common organisms of CAP. Interestingly enough, the most common etiologies of CAP vary depending on the severity of illness (Mandell et al., 2007). This is an example of how risk stratification and acuity of illness can help guide treatment.

Bacterial or atypical bacterial pathogens commonly responsible for CAP in the patient stable enough to be treated outpatient include *Streptococcus pneumoniae, Haemophilus influenzae, Chlamydia pneumoniae, Moraxella catarrhalis,* and *Mycoplasma pneumoniae* (Brashers, 2008). Respiratory viruses also cause CAP in the outpatient population and include viruses such as influenza A and B, adenovirus, respiratory syncytial virus, and parainfluenza virus (Mandell et al., 2007; Miskovich-Riddle & Keresztes, 2006).

Individuals with CAP, ill enough to require hospitalization in the nonintensive care unit (ICU) environment, commonly are infected with similar organisms as the outpatient with CAP with the addition of *Legionella pneumophila* and anaerobes associated with aspiration as an etiology for illness. Severely ill CAP patients requiring
ICU admission are most commonly infected with *S. pneumoniae*, *H. influenzae*, *Staphylococcus aureus*, *L. pneumophila* and other gram negative bacilli (Mandell et al., 2007; Plouffe & Martin, 2008). Patient disposition based on severity of illness can help with the initiation and subsequent treatment of patients hospitalized with CAP.

**Clinical presentation**

Patients with CAP are often seen first by a primary care, urgent care, or emergency room provider. Symptoms can be classic in presentation and include cough, fever, chest pain, tachypnea, and dyspnea or less obvious in presentation with no respiratory symptoms and only mental status changes or unexplained arthralgias and myalgias. Severe cases may present with altered level of consciousness, sepsis, and respiratory failure. Additional constitutional symptoms may include fatigue, headache, malaise, nausea, and vomiting (Mandell et al., 2007; Plouffe & Martin, 2008).

**Diagnosis/evaluation**

**History**

A thorough history is essential in the diagnosis and management of CAP. Initial components of the history should explore the onset of presenting symptoms, location of the primary symptoms, duration of illness, character of cough, sputum, and pain, aggravating factors, relieving factors, treatments tried, and a patient rating of severity of illness or pain. Alert symptoms of potential CAP are complaints of dyspnea, orthopnea, fatigue, weakness, and confusion. Negative symptoms and test results can be as helpful as positive ones when evaluating for CAP versus other differential diseases. The presence or absence of chest pain, weight gain, edema, abdominal pain, dysuria, hematuria, and neurological symptoms can help the clinician arrive at the appropriate diagnosis.

Patient history is essential to arrive at the diagnosis of CAP. Historical data influence components of the physical assessment, development of the diagnosis, and creation of a holistic treatment plan specific to each patient. A list of current medications and allergies should also be elicited, including prescriptions, over the counter therapies, and herbal remedies. Additional historical elements not to be missed include the past medical, family, social, and health maintenance histories.

Past medical history should survey for chronic diseases, such as autoimmune illnesses, cancer, chronic respiratory illnesses, recent acute illnesses, and recent hospitalizations. The family history should include an assessment of any acute illnesses in the household, chronic respiratory diseases (e.g., tuberculosis) and chronic tobacco use in the home. Elements of the social history should include patient use of tobacco, alcohol, occupational exposure to lung irritants, and narcotic or illicit drug use. To ascertain if this is a case of CAP versus other classification of pneumonia, it is also important to assess for place of residence be it home, institution, assisted living facility, nursing home, or homeless shelter. Health maintenance history should include immunization status of influenza and pneumovax shots.

**Physical exam**

The physical exam will give further clues to the diagnosis of CAP or acute illnesses that may have contributed to the development of CAP. Upon initial observation confusion, weakness, shortness of breath may be noted. Key exam components should include but are not limited to, vital signs, a general survey, head, eyes, ears, nose, throat (HEENT), neck, chest, respiratory, cardiac, skin, and mental status. CAP may affect vital signs including fever, tachypnea, tachycardia, hypotension, and hypoxia. A general survey may reflect the appearance of fatigue, illness, or impaired level of consciousness.

A thorough physical assessment will give further evidence of pneumonia. The HEENT exam may show signs of upper respiratory infections, such as otitis media, nasal congestion or discharge, sinus tenderness, ocular discharge, and pharyngeal erythema with or without exudate. Evidence of respiratory distress may include mouth breathing, nasal flaring, or grunting. Dehydration, a common complication of acute illness can be evidenced on the eyes, ears, nose and throat exam with dullness to the eyes, dry lips, and buccal mucosa.

CAP is an illness that can affect many systems of the body. Abnormalities in the neck exam of the CAP patient may include lymphadenopathy. Carotid bruits and jugular venous distension should be assessed to rule out other disease processes responsible for or complicated by the presenting symptoms. The chest exam may reveal intercostal retractions or accessory muscle use. Dullness to percussion, increased tactile fremitus, egophony, bronchophony, prominent whispered pectoriloquy, and chest tenderness can help identify areas of lung consolidation. Findings on auscultation may include diminished lung sounds, wheezes, crackles, rhonchi or a combination of these adventitious sounds. An oxygen saturation level can detect hypoxia, is an indicator of severity of illness, and should be obtained as part of the vital signs.

Additional systems affected by CAP may include cardiac, skin, and mental status. The cardiac exam may show tachycardia and evidence of poor perfusion with prolonged capillary refill. The heart sounds and point of
maximal impulse should be noted as they can indicate congestive heart failure, a differential of CAP. The abdomen should be assessed as should the musculoskeletal and neurological system to further gain information on the general condition of the patient. Skin may have poor turgor and tenting because of dehydration in addition to cyanosis from hypoxia. Mental status can show evidence of confusion because of hypoxia.

Differential diagnoses

The presentation of CAP can be straightforward to subtle and complex. Other differential diagnoses to consider when confronted with the signs and symptoms of CAP include exacerbation of chronic obstructive pulmonary disease, central nervous system disease, inhalation injury, reactive airway disease, bronchitis, influenza, lung cancer, pulmonary embolus, congestive heart failure, tuberculosis, myocardial infarction, atelectasis, or empyema (Chesnutt & Prendergast, 2011; Plouffe & Martin, 2008). Table 1 compares common differentials when considering the diagnosis of CAP. It is the thorough history, physical, and diagnostic findings that help differentiate CAP from other potential illnesses.

Diagnostic tests

When CAP is suspected, a chest radiograph should be obtained when possible, as infiltrates on chest x-ray confirms the diagnosis of pneumonia. The type of infiltrate can help the clinician differentiate between viral and bacterial pneumonia. Interstitial infiltrates are most common in viral pneumonia, while alveolar infiltrates suggest bacterial pneumonia. As viral and bacterial pneumonia can occur simultaneously, the clinician may see a variety of presentations on chest x-ray and will have to rely on assessment findings, clinical judgment, and risk stratification to determine best treatment (Ruuskanen, Lahti, Jennings, & Murdoch, 2011).

Chest x-ray can also help evaluate for resolution of infection in the identification of other lung disease or underlying illnesses, such as neoplasm. Most radiologic evidence of CAP resolves within 6 weeks (Chesnutt & Prendergast, 2011). According to Mandell et al. (2007), CAP should be followed up with a repeat chest x-ray 12 weeks after symptom resolution to rule out underlying clinical pathology.

A complete blood count should be obtained on all patients suspected of CAP regardless of severity. According to Plouffe and Martin (2008), patients determined to have mild CAP treated at home need no additional laboratory data. Sputum cultures can be useful in identifying causative organisms of CAP but should only be collected if the sample is of good quality. This means the sputum specimen should be from a deep cough and cultured within 2 h of being obtained. Culture of aspirate from thoracentesis should be obtained if found to be significant on chest x-ray. Blood cultures are not necessary for patients stable enough to be treated on an outpatient basis. Inpatients with CAP should not only have blood cultures, but should also have urine tested for the antigens of L. pneumophila and S. pneumoniae (File, 2011; Mandell et al., 2007).

Risk stratification

Disposition of patients identified with CAP can be a challenge. The literature shows a significant number of CAP cases can be successfully treated in the outpatient setting. “Physicians tend to overestimate a patient’s risk of death; therefore, many low-risk patients who could be safely treated as outpatients are admitted for more costly inpatient care” (Lutfiyya et al., 2006, p. 455).

Guidelines for outpatient therapy resulted in a need for validated clinical tools to help clinicians decide on inpatient or outpatient CAP treatment. In order to help providers identify patients who are able to be treated empirically on an outpatient basis, the pneumonia severity index (PSI) and CURB-65 scale were created. CURB-65 is a modification of the British Thoracic Society rule and stands for confusion, uremia, respiratory rate, blood pressure, and 65 years of age. These tools are designed to aid in the risk stratification of patients with CAP but are not intended to replace clinical judgment (Ebell, 2006).

Pneumonia severity index. The PSI is a risk model created by investigators of a large CAP cohort study to classify risk for patients with CAP. As a tool, the PSI has been validated in several large investigations in settings, such as nursing homes, emergency departments, and community hospitals. The PSI has been found to be more effective in determining the severity of pneumonia and the need for hospitalization than the CURB-65. PSI is well supported in the literature and should be considered as a potential tool for risk stratification when determining which patients are appropriate for treatment in the outpatient setting (Lutfiyya et al., 2006; Nazarian et al., 2009).

The PSI evaluates risk by assessing individual patient characteristics, including demographics, comorbidities, physical exam findings, laboratory, and radiographic findings. Demographic risks include gender and nursing home residency. Scored comorbidities include neoplasm, liver disease, heart failure, stroke, and renal failure. Physical exam findings pertinent to the PSI scoring system include mental status, respiratory rate, systolic
### Table 1 Differentials for CAP

| Differential                  | Positive findings                          | Negative findings                          |
|------------------------------|--------------------------------------------|--------------------------------------------|
| Pneumonia                    | Fever/chills                               | Cardiac enzymes                            |
|                              | Shortness of breath                        | computed tomography (CT) of brain          |
|                              | Tachypnea                                  | D dimer                                    |
|                              | Cough                                      | Hypotension                                |
|                              | Tachycardia                                |                                            |
|                              | Rales                                      |                                            |
|                              | Pleuritic chest pain                       |                                            |
|                              | Dyspnea                                    |                                            |
|                              | Sputum production                          |                                            |
|                              | Nausea                                     |                                            |
|                              | Vomiting                                   |                                            |
|                              | Diarrhea                                   |                                            |
|                              | Mental status changes                      |                                            |
|                              | Chest pain                                 |                                            |
|                              | Leukocytosis with leftward shift           |                                            |
|                              | Positive blood cultures                    |                                            |
|                              | Infiltrate on chest x-ray (CXR)             |                                            |
|                              | Hypoxemia                                  |                                            |
|                              | Acute exacerbation of pulmonary disease    |                                            |
|                              | Increased cough                            | No infiltrate on CXR                        |
|                              | Sputum production increase                 | Hypotension                                |
|                              | Dyspnea                                    |                                            |
|                              | Tachypnea                                  |                                            |
|                              | Hypercapnea                                |                                            |
|                              | Changes in mental status                   |                                            |
|                              | CXR chronic changes                        |                                            |
|                              | Dyspnea                                    |                                            |
|                              | Rales                                      |                                            |
|                              | Peripheral edema                           |                                            |
|                              | Mental status changes                      |                                            |
|                              | Decreased urine output                     |                                            |
|                              | Weight gain                                |                                            |
|                              | Worsening renal function                   |                                            |
|                              | Dyspnea                                    |                                            |
|                              | Tachypnea                                  |                                            |
|                              | Hypoxemia                                  |                                            |
|                              | Jugular venous distention                  |                                            |
|                              | Electrolyte disturbance                    |                                            |

### Table 1 (Continued)

| Differential                  | Positive findings                          | Negative findings                          |
|------------------------------|--------------------------------------------|--------------------------------------------|
| Pulmonary embolus            | Hypoxemia                                  | Sputum culture                            |
|                              | Hypoxemia                                  | negative                                  |
|                              | Hypocapnia                                 | No fever                                  |
|                              | Respiratory alkalosis                      | Negative cultures and gram stains         |
|                              | Shortness of breath                        | Purulent sputum                            |
|                              | Infiltrate                                 | No fever spikes                            |
|                              | computed tomography (CTPA) of lungs        |                                            |
|                              | stocked pulmonary angiogram                |                                            |
|                              | Elevated B type                            | Lab changes                                |
|                              | Hypotenion                                 | Increase in sputum production             |
|                              | Elevation B type                           |                                            |
|                              | Hypotension                                |                                            |
|                              | Myalgia/malaise                            | Dyspnea                                   |
|                              | Weakness                                   | Hypoxemia                                 |
|                              | Cough non productive                       | Hyper/hypocapnia                           |
|                              | Sore throat                                | Hypotension                               |
|                              | Nasal discharge                            | Tachycardia                               |
|                              | Mild lymphadenopathy                       |                                            |
|                              | Positive influenza nasal swab              |                                            |
|                              | Fever                                      | Infiltrates                               |
|                              | Headache                                   | Tachypnea                                 |
|                              | Myalgia/malaise                            | Dyspnea                                   |
|                              | Weakness                                   | Hypoxemia                                 |
|                              | Cough non productive                       | Hyper/hypocapnia                           |
|                              | Sore throat                                | Hypotension                               |
|                              | Nasal discharge                            | Tachycardia                               |
|                              | Mild lymphadenopathy                       |                                            |
|                              | Positive influenza nasal swab              |                                            |
|                              | Myocardial infarction                      |                                            |
|                              | Hypotension                                | Fever                                     |
|                              | Clear lung sounds                          | Cough                                     |
|                              | Rise or fall of cardiac                    | Malaise                                   |
|                              | Biomarkers                                 | Sore throat                               |
|                              | Electrocardiogram (ECG) changes            | Nasal discharge                           |
|                              | Chest pain                                 | Lymphadenopathy                           |
|                              | Imaging evidence of wall                   | Infiltrates                               |
|                              | motion abnormalities                       |                                            |

Source: Bartlett (2012); Colucci (2012); Dolin (2012); Reeder (2012); Stoller (2012); Taylor-Thompson (2012).

Blood pressure, temperature, and pulse. Laboratory and radiographic findings assessed with the PSI include arterial pH, blood urea nitrogen, sodium, glucose, hematocrit, partial pressure of arterial oxygen, and pleural effusion on x-ray (Ebell, 2006).

The PSI risk classification serves as a guide for outpatient versus inpatient treatment. PSI scoring is placed in a risk class of I–V based on the number of points accumulated. Patients with a risk classification of I or II (<51–70 points) are considered safe to treat on an outpatient basis. Those with a risk class of III (71–90 points) can be managed outpatient but will need close clinical observation by their NP. Patients with a PSI score of IV and V (91 to >130 points) are high-risk patients and need to be hospitalized for pneumonia treatment (Ebell, 2006; Luftiyya et al., 2006).

Carratala et al. (2005) studied the outcomes of patients with a PSI of class II or III and found no statistically significant difference between the patient’s outcomes when treated inpatient versus outpatient. “Although it is assumed that inpatient care leads to better outcomes, our study shows that hospitalization places patients at risk for complications such as phlebitis or pulmonary embolism”
(Carratala et al., 2005, p. 171). In another study of the PSI, Labarere et al. (2006) showed low risk individuals with CAP treated as outpatients returned to usual activities and work 6–9 days sooner than low risk patients who had been hospitalized.

CURB-65. The CURB-65 is a quicker assessment and much more useful tool at the point of care because of simplicity of use. “CURB-65 includes only five variables compared with the 20 in the PSI. The CURB-65 also provides a four-variable substitute for use where blood testing is not immediately available” (Ebrell, 2006, p. 41). Clinical factors scored by the CURB-65 include confusion, BUN, respiratory rate, blood pressure, and age (Ebrell, 2006).

Curb-65 scores range from 0 to 5. Individuals with a score of 0–1 are considered low risk and should be treated outpatient. A score of 2 may require close outpatient observation or short inpatient hospitalization depending on clinician judgment and patient circumstances. Patients with psychosocial circumstances such as inability to follow directions, lack of social support, or transportation may benefit from a brief inpatient stay. Patients with a score of 3 or greater are candidates for inpatient hospitalization (Ebrell, 2006).

According to Anada-Rajah et al. (2008), the PSI is more accurate in identifying patients with severe illness. This includes those at highest risk for mortality within 30 days, and those who may need ICU admission, such as the elderly. The CURB-65 is a good tool in the outpatient setting when laboratory data are not immediately available. Inpatient treatment of CAP is expensive compared to outpatient treatment. There would be a significant cost savings in healthcare, if providers appropriately utilize the PSI or CURB-65 severity scales to identify suitable patients for outpatient treatment. The PSI and CURB-65 assessment tools can be accessed at http://www.aafp.org/fpm/20060400/41outp.html (Ebrell, 2006).

**Outpatient plan/management**

**Pharmacologic management**

Outpatient treatment for CAP includes the use of empirical antibiotics for the most common pathogens identified above in the pathogenesis section. In 2007, guidelines were developed with a consensus from both the American Thoracic Society (ATS) and the Infectious Disease Society of America (IDSA) for the management of CAP. These guidelines include diagnosis, treatment, and follow-up recommendations.

The emergence of antibiotic resistance has stressed the importance of antimicrobial stewardship and evidence-based prescribing for many infectious diseases including those responsible for CAP (Tamma & Cosgrove, 2011). In an effort to reduce the incidence of antibiotic resistance and adverse side effects from antibiotics, shorter courses of antibiotic therapy, as little as 5 days, are recommended. The literature shows that there is no clinical difference in patient outcomes between short and long course therapy for CAP (Siempos, Dimopoulos, & Falagas, 2009).

Patients need to be clinically stable for short course antibiotic therapy. This includes being afebrile for 48–72 h of the last 5 days of therapy, heart rate less than 100, systolic blood pressure greater than 90, respiratory rate less than 24, oxygen saturation greater than 90%, and a normal mental status. If the patient is not clinically improved at day 5, antimicrobial therapy should continue for an additional 2 days (Mandell et al., 2007).

Shorter antibiotic therapy has been found to reduce the incidence of adverse events such as Clostridium difficile–related diarrhea and have the same success rate for patients with mild to moderate CAP (Li, Winston, Moore, & Bent, 2007).

Severity of illness, clinical symptoms, age, local infectious patterns, previous antibiotic exposure, comorbidities, and cost-effectiveness must be analyzed when choosing the best antibiotic therapy (Lutfiyaa et al., 2006). Current research for empiric antibiotic treatment is based on medical history. Healthy patients with low risk for drug-resistant Staphylococcus pneumoniae (DRSP) should be treated differently than individuals with comorbidities, at risk for DRSP or who live in regions of the country with a high rate of macrolide resistance, see Table 2 (Mandell et al., 2007).

**Antibiotic and antiviral treatment.** The IDSA/ATS guideline summarized in Table 2 includes the most recent recommendations for the treatment of CAP with empiric antibiotics. Macrolides are the first line treatment for healthy individuals with no antibiotic use in the last 3 months. Doxycycline can also be used in this low-risk population as an alternative to a macrolide. These antibiotic classifications will cover the common atypical organisms M. pneumoniae and Chlamydia pneumoniae (Mandell et al., 2007). “The use of fluoroquinolones to treat ambulatory patients with CAP without comorbid conditions, risk factors for DRSP, or recent antimicrobial use is discouraged because of concern that widespread use may lead to the development of fluoroquinolone resistance” (Mandell et al., 2007, p. S484).

Patients that have taken antibiotics within the last 3 months or have other comorbidities (chronic heart, lung, liver, or renal disease; diabetes; asplenia; alcoholism, malignancy, or are immunocompromised) should have treatment that includes fluoroquinolones (gemifloxacin, moxifloxacin, levofloxacin) or beta-lactams (high-dose amoxicillin or amoxicillin-clavulanate) plus a macrolide.
Table 2 Outpatient empiric treatment of CAP

| Patient health status                                                                 | Treatment options                                                                 |
|--------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------|
| Healthy, no use of antimicrobials in the past 3 months                                | Macrolide (azithromycin, clarithromycin, erythromycin)                            |
|                                                                                      | Or                                                                                |
|                                                                                      | Doxycycline                                                                      |
| Comorbidities (antibiotics within last 3 months, chronic heart, lung, liver or renal disease, diabetes, asplenia, alcoholism, malignancy, or immunocompromised) | Respiratory fluoroquinolone (gemifloxacin, moxifloxacin, levofloxacin [750 mg]) |
|                                                                                      | Or                                                                                |
|                                                                                      | Beta-lactam (high dose amoxicillin 1 g tid or amoxicillin-clavulanate 2 g po bid) plus macrolide or doxycycline |
| Region with high rate (>25%) of high level (>16 μg/mL) macrolide resistant Streptococcus pneumoniae | Respiratory fluoroquinolone (gemifloxacin, moxifloxacin, levofloxacin [750 mg]) |
|                                                                                      | OR                                                                               |
|                                                                                      | Beta-lactam (high dose amoxicillin 1 g tid or amoxicillin-clavulanate 2 g po bid) plus macrolide or doxycycline |

*Alternative ceftriaxone, cefpodoxime, and cefuroxime (500 mg two times daily), doxycycline is an alternative to the macrolide.

*High dose to cover drug-resistant Staphylococcus pneumoniae (DRSP), high-risk patients include <2 and >65 beta-lactam therapy within the last 3 months, alcoholism, comorbidities, immunocompromised, or exposure to child in day care.

Source: (Mandell et. al., 2007).

(Azithromycin) or doxycycline. The high-dose amoxicillin is used for patient at high risk for DRSP. Populations at risk include those <2 and >65 years; family members or employees exposed to children in day care settings; recipients of beta-lactam therapy within the last 3 months; and persons with alcoholism, comorbidities, or immunocompromise. Individuals who live in a region with a high rate (greater than 25%) or high level (>16 μg/mL) of macrolide resistant S. pneumoniae should also be treated with respiratory fluoroquinolones or beta-lactams plus a macrolide or doxycycline. Cephalosporins (ceftriaxone, cefpodoxime, and cefuroxime) may be used as an alternative to the beta-lactams or high-dose amoxicillin if amoxicillin allergy is present (Mandell et al., 2007).

To identify if the local clinician is in a region with high rates of drug resistance, the NP should contact their local laboratory to obtain current antibiogram information about the resistant rates in their own communities. An antibiogram, “is a chart indicating which organisms are demonstrating resistance in a particular locale” (Hannigan, Tolman, & Larson, 2012, p. 325).

Antivirals have been researched since the 2009 H1N1 outbreak and have been found to decrease mortality rates when initiated quickly for patients with viral pneumonia or combined viral–bacterial pneumonia (CDC, 2011a; Minakami et al., 2011; Yang et al., 2012). Although pediatric guidelines have included the use of antivirals for the treatment of CAP, there are no current guidelines for the use of antivirals with adult populations. As more evidence becomes available, antivirals such as oseltamivir and/or zanamivir may become standard of care for initial treatment of CAP when suspicion of viral pneumonia is high. Even with the use of antivirals, the NP will have to carefully assess for symptoms of secondary bacterial pneumonia, so antibiotics can be initiated as appropriate. Table 3 outlines antimicrobial selection based on frequent etiologies of pneumonia including viral infections.

Supportive pharmacological and symptom management. Antimicrobials are not the only treatment for CAP. Antipyretics such as ibuprofen and acetaminophen can be used for fever and malaise associated with CAP. Systemic corticosteroids can be used for the patient with reactive airway symptoms because of CAP. Steroid treatment of individuals with CAP without reactive airway symptoms is controversial and lacks evidence for efficacy to date (Brown & Dean, 2011).

Additional therapies may include the use of bronchodilators, cough suppressants, and adequate nutrition and hydration. Bronchodilators may need to be used to open up airways inflamed by cytokine release with CAP (Restrepo & Anzueto, 2009). Prescription cough suppressants are generally not recommended, but can be used judiciously in individuals unable to sleep because of significant coughing. Encouragement of adequate fluid intake is also important to avoid dehydration and facilitate clearance of secretions. Good nutrition is also recommended to provide the adequate nutrients required to facilitate healing from CAP and to support the body’s immune response.
### Table 3  CAP treatment by etiology

| Possible etiology | Risk groups                                                                 | Medication management                                                                 |
|-------------------|-----------------------------------------------------------------------------|---------------------------------------------------------------------------------------|
| *Streptococcus pneumoniae* PCN susceptible | Smokers, HIV infection, Influenza infection, injection drug users | First line: penicillin G, amoxicillin, Alternatives: doxycycline, macrolide (beware of macrolide resistance), cephalosporin (oral or parenteral), clindamycin, respiratory fluoroquinolone. |
| *S. pneumoniae* PCN resistant some macrolide and DRSP |  | DRSP risk: treatment based on sensitivity testing—cefotaxime, ceftriaxone, respiratory fluoroquinolone, vancomycin, linezolid, high-dose amoxicillin. |
| *Haemophilus influenzae* non-beta-lactamase producing | *H. influenzae* is an aerobic bacillus can be encapsulated and nonencapsulated. Encapsulated type B (HiB) is very virulent but with vaccination incidences have decreased | First line: amoxicillin, Alternative: respiratory fluoroquinolone, doxycycline, azithromycin, clarithromycin. |
| *H. influenzae* beta-lactamase producing | Comorbidities                                                                 | First line: second- or third-generation cephalosporin, amoxicillin/clavulanate, Alternative: respiratory fluoroquinolone, doxycycline, azithromycin, clarithromycin. |
| *Moraxella catarrhalis* (PCN and beta-lactam antibiotic resistance) | Respiratory fluoroquinolones                                                                 | Respiratory fluoroquinolones                                                                 |
| *Group A Streptococcus* | Recent viral illness such as influenza. Close contact of other with *Streptococcus* infections | First line: antistaphylococcal penicillin, Alternatives: cefazolin, clindamycin. |
| *Staphylococcus aureus* (methicillin susceptible) | *S. aureus* may cause CAP with those infected with influenza (H1N1 or seasonal flu) | First line: vancomycin, linezolid, Alternatives: trimethoprim–sulfamethoxazole. |
| *S. aureus* (methicillin resistant/ susceptible to clindamycin) | Patients that are intravenous drug abusers (IVDAs) or have other debilitations | First line: vancomycin, linezolid, Alternatives: trimethoprim–sulfamethoxazole. |
| *S. aureus* (methicillin resistant/resistant to clindamycin) |  | First line: vancomycin, linezolid, Alternatives: trimethoprim–sulfamethoxazole. |
| Methicillin-resistant *Staphylococcus aureus* (MRSA) |  | First line: antistaphylococcal penicillin, Alternatives: cefazolin, clindamycin. |
| *Klebsiella pneumoniae* |  | First line: antistaphylococcal penicillin, Alternatives: cefazolin, clindamycin. |
| *Pseudomonas aeruginosa* | Gram-negative: alcoholism, pregnant women coinfected with H1N1, diabetes, or COPD | High resistance to ampicillin. Cephalosporins, aminoglycosides, and fluoroquinolones. |
|  | Gram-negative pneumonias occur most often in debilitated individuals, chronic oral steroid use, immunocompromised, underlying bronchopulmonary disease, patients with recent hospitalization, frequent antibiotic use, smokers | First line: antipseudomonal beta-lactam (piperacillin/tazobactam, cefepime, imipenem, or meropenem) plus ciprofloxacin, levofloxacin, or aminoglycoside. |
|  | Alternative: aminoglycoside plus ciprofloxacin or levofloxacin | Alternative: beta-lactam plus aminoglycoside plus azithromycin. |
|  | Alternative: beta-lactam plus aminoglycoside plus fluoroquinolone | Alternative: beta-lactam plus aminoglycoside plus fluoroquinolone. |
|  | PCN allergy: aztreonam for beta-lactam | Alternative: respiratory fluoroquinolone. |
|  | No cell wall so PCN and cephalosporin are not effective | Alternatives: respiratory fluoroquinolone. |

(Continued)
Table 3 (Continued)

| Possible etiology | Risk groups | Medication management |
|-------------------|-------------|-----------------------|
| Nonzoonotic atypical: *Chlamydia pneumoniae* | COPD and/or smoker | First line: macrolide, tetracycline_alternative: respiratory fluoroquinolone |
| Nonzoonotic atypical: *Legionella* species | Hotel or cruise ship travel, smokers | First line: fluoroquinolone, azithromycin_alternative: doxycycline |
| Viral respiratory illness/pneumonia | Influenza outbreak within a community | If available, use information regarding the strain of flu for selection of antivirals for treatment/prevention of viral pneumonia |
| Viral respiratory illness/pneumonia (influenza types A and B, H1N1, RSV, adenovirus, parainfluenza, rhinoviruses, SARS—coronaviruses, human metapneumovirus) | Viral pneumonia is more common in young children and elderly | Early treatment of influenza infection in ambulatory adults with inhaled zanamivir or oral oseltamivir |
| | | Influenza A and B: neuraminidase inhibitors (oseltamivir, zanamivir) for early treatment. |
| | | Seasonal H1N1 shown resistance to oseltamivir, recommended using zanamivir, amantadine, or rimantadine |
| | | Seasonal H3N2 resistant to amantadine and rimantadine, use zanamivir or combination of oseltamivir and rimantadine |
| | | 2009 H1N1 virus is still susceptible to neuraminidase inhibitors. Oseltamivir or zanamivir is recommended treatment, some resistance to oseltamivir has been found with immunocompromised populations |
| | | Respiratory syncytial virus: for severe illness ribavirin (inhaled or IV) |
| | | Adenovirus: for severe illness cidofovir |
| | | Varicella-zoster pneumonia: IV acyclovir |
| | | Hantavirus: supportive, IV ribavirin |
| | | Human metapneumovirus: for severe illness IV ribavirin |
| | | Parainfluenza: for severe infection ribavirin |
| | | Herpes simples virus: acyclovir |
| | | Coronavirus (SARS): lopinavir/ritonavir |
| | | No studies indicating antivirals helpful for infections with rhinovirus |
| | | Recommended treatment: cefotaxime, ceftriaxone, and respiratory fluoroquinolones |
| | | CA-MRSA suspicion (confirmed laboratory or clinical presentation): vancomycin, linezolid, or other agents for CA-MRSA |

Sources: Bradley et al. (2011); CDC (2011); Cunha (2012); Lin, Jeng, Chen, and Fung (2010); Mandell et al. (2007); Mosenifar et al. (2011); Murphy and Parameswaren (2009); Niederman (2010); Okimoto et al. (2011); Ruuskanen et al. (2011).

Follow-up

Follow-up for the outpatient with CAP is generally in 48–72 h after initial diagnosis and then again in 2–3 weeks. Patients should always be instructed to be seen sooner should any complications or concerns develop. Individuals who are tobacco smokers should also been seen again in 6–12 weeks for repeat evaluation and repeat chest x-ray to rule out any underlying pathology that could have contributed to CAP.
Health promotion/disease prevention

Health promotion and disease prevention strategies, such as smoking cessation, immunizations, and judicious use of antibiotics have been successful in decreasing the mortality and hospitalization rates of CAP. According to the CDC (2010), CAP decreased from the seventh leading cause of death in 2006 to the ninth leading cause of death in the United States in 2009.

A retrospective review of the data available on HCUPnet shows a decrease in the rate of hospitalizations for CAP by 60,005 persons annually when comparing the data reported in 1997 to that available in 2007 (Agency for Healthcare Research and Quality, 2009). The healthcare utilization project (HCUP) database is a compilation of national data from state data organizations, hospital associations, private data organizations, and the Federal government. HCUP includes a large collection of longitudinal hospital care data in the United States, including data on hospitalization rates for pneumonia.

Zagaria (2010) discusses the lack of clinical evidence that smoking cessation reduces pneumonia rates; but, understanding that smoking alone increases risk of death makes it a logical conclusion that smoking cessation does reduce risk of CAP. Smoking cessation is such an important risk reduction strategy for pneumonia and other diseases that it is a quality measure used by the Joint Commission and the Centers for Medicare and Medicaid Services (Shorr & Owens, 2009). It is also the focus of the Patient Protection and Portable Care Act signed into law by President Obama in May, 2010 (Franken, 2010).

The new healthcare act not only increases patient access to health care, but it also focuses on health care reform, with an emphasis on disease prevention and health promotion like never before in the United States. According to Franken (2010), “health reform will work on multiple levels to prevent illness, intervene early when risk of illness occurs, help those with chronic illness from becoming sicker, and generally keep Americans healthier, longer” (p. S83). A focus on reducing preventable deaths because of tobacco use is just one of the healthcare reform strategies now possible because of adequate funding from this legislation (Franken, 2010).

The impact of tobacco use has been well studied and documented over the years, as have cessation strategies. Cessation strategies studied and found to have increased success in getting patients to stop smoking include healthcare provider screenings for tobacco use through the use of electronic medical record (EMR) reminder systems or health maintenance flow sheets, followed by support and patient education. Identifying patients as tobacco users is the first step in addressing this serious risk factor with patients during routine visits (Atherton, Car, & Meyer, 2010; Boyle, Solberg, & Fiore, 2010; Hesse, 2010).

Healthcare providers can provide education, support, and pharmacological interventions to assist patients with smoking cessation. The new era of healthcare reform and support for EMRs makes cessation strategies for the NP striving to adhere to best practice more efficient than in the past. An example of this is the enrollment of identified tobacco users in e-mail programs that offer support, cessation resources, service invitations, and encouragement for patients contemplating or committed to smoking cessation (Atherton et al., 2010; Boyle et al., 2010; Hesse, 2010).

The pneumovax vaccine is a cost-effective means to protect persons over the age of 65 and those with high-risk concurrent diseases from pneumonia (Counts & Rehm, 2011). Pneumonia vaccinations should be offered to all adults 65 years and older and to individuals considered high risk for pneumonia. High-risk individuals include those with chronic lung disease, diabetes, heart disease, and those with a compromised immune system. If the pneumonia vaccine is administered to a high-risk individual or to a patient before the age of 65 it should be boosted once 5 years after the initial immunization to prevent waning immunity (CDC, 2012).

Current statistics for pneumonia vaccinations show 59% of persons over age 65 are immunized (CDC, 2012). According to Rosenberg et al. (2010), the pneumococcal vaccine has reduced the incidence of invasive pneumococcal disease by 77% from 2000 to 2005 in children less than 2 years. There was also an additional herd effect benefiting unvaccinated individuals because of the decrease in nasopharyngeal colonization. The vaccine can also reduce the incidence with older adults, especially women, and the transmission from children to adults normally seen during the winter months (Walter, Taylor, Dowell, Mathis, & Moore, 2009).

The influenza vaccine has been shown to prevent the flu in 70%–90% of individuals younger than 65 years of age and should be administered annually (Miller & Drinka, 2007). Miller and Drinka (2007) found, “the influenza vaccine prevents 50%–60% of hospitalizations and pneumonia and is 80% effective in preventing death in nursing home residents” (p. 47). Influenza and pneumonia are the seventh leading cause of death for those over 65 years of age (American Lung Association, 2010). Because of this increased risk, a high dose influenza vaccine was offered for adults 65 years and older for the 2011–2012 flu season (CDC, 2011b).

Clinicians are now being encouraged to vaccinate their populations earlier in the season as the immunity has been found to last throughout the entire season.
Vaccinating patients in August and September allows herd immunity to build up by the time infections begin. Egg allergy restrictions have also been lessened and only those patients with true anaphylaxis should avoid the influenza shot. Patients without a history of anaphylaxis because of eggs can be administered the vaccine and observed for 30 min in the clinic setting as long as the clinic is equipped to handle anaphylactic emergencies. The inhaled nasal influenza vaccine should still be avoided in individuals with egg allergy (CDC, 2011b).

Several research initiatives have investigated health promotion strategies to increase immunization rates for the pneumonia and influenza vaccines. Standing immunization orders, EMR reminder systems, health maintenance flow sheets, and patient access to personal health records are all ways to increase immunization rates and therefore decrease the risk of CAP. Providers that write standing immunization orders allow nurses to immunize individual patients that present to the practice for a flu or pneumonia immunizations without the need or expense of a formal office visit. This allows nurses to offer immunization clinics at their place of work and in the community (Zimmerman et al., 2011).

EMR reminder systems prompt both nursing and healthcare providers at the point of care regarding the need for influenza and pneumonia immunizations. Individuals lacking immunizations can be vaccinated immediately. These systems can also be used to provide reminder mailings to patients who are due for immunizations. Taking it a step further and allowing patients access to their own healthcare summary through patient portals allows them to evaluate their own health promotion and disease prevention needs (Atherton et al., 2010; Caligtan & Dykes, 2011; Dexheimer et al., 2011; Nowalk, Zimmerman, & Fegali, 2004). The advent of the EMR, reminder systems, patient support, and standing orders are all useful tools proven to increase immunization rates and improve smoking cessation rates in practice.

Implications for practice

Even in the presence of increased antibiotic resistance, immunizations, risk stratification, and appropriate antibiotic use have contributed to a decline in the number of hospitalizations and deaths related to pneumonia from 1997 to 2007. The literature shows the key to decreasing hospital admissions and overall cost of CAP treatment is identifying patients at low risk of mortality, thereby safely treating them on an outpatient basis.

Determination of hospitalization for CAP is difficult with the multiple factors both clinical and social that must be considered. There is an increased risk of mortality associated with “respiratory rates greater than 30, diastolic hypotension, and an elevated blood urea nitrogen level” (Johnstone, Majumdar, & Marrie, 2008, p. 215). Risk estimates vary widely by providers and it is thought that variations and systemic overestimates of mortality affect hospital admission rates despite CAP incidence (Johnstone et al., 2008). NPs are initial responders who can help identify, prevent, and treat CAP. These efforts will continue to support the downward trend of hospital admissions for those at low risk with CAP.

Unfortunately, hospitalization for bacterial pneumonia continues to consume large amounts of healthcare dollars annually in spite of decreased hospitalization rates and length of stay. The data suggest there continues to be room for avoidance of this deadly disease through aggressive outpatient prevention and management strategies. NPs need to evaluate the severity of pneumonia by using tools such as the CURB-65 or PSI and use sound clinical judgment of findings to appropriately determine candidates for outpatient versus inpatient treatment.

Using the 2007 established guidelines from the ATS/IDSA will promote effective therapy using evidence-based research and will allow for quicker resolution of symptoms and avoidance of costly hospital admissions. Pneumonia prevention, identification, and treatment are often initiated by a family, urgent care, or emergency NP. Providers in this role have the ability to further reduce healthcare costs, decrease incidence of CAP, and improve patient outcomes for individuals with CAP through the use of risk stratification, and appropriate antibiotic use.

Conclusion

As a nation, we have improved hospitalization rates for CAP over a decade using the strategies of risk analysis, health promotion, and judicious antibiotic use. Current healthcare reform, with a stronger focus on disease prevention supports healthcare providers in their efforts to continue with the downward trend in hospitalizations related to CAP. Outpatient treatment, when appropriate, is cost-effective and has shown to return the patient back to normal activities much sooner. NPs are often in the frontline of care and have the means to prevent and treat CAP, thus, improving patient outcomes and healthcare costs.

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