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Nosocomial Pneumonia

Burke A Cunha, MD, Professor of Medicine, State University of New York School of Medicine at Stony Brook; Chief, Infectious Disease Division, Winthrop-University Hospital

Updated: May 28, 2009

Introduction

Background

According to recent American Thoracic Society (ATS) guidelines, nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP]) is defined as pneumonia that occurs more than 48 hours after admission but that was not incubating at the time of admission. Ventilator-associated pneumonia (VAP) is defined as pneumonia that occurs after 48-72 hours of endotracheal intubation. Nosocomial pneumonia is the second-most-common nosocomial infection and is usually bacterial in origin. The disease adds significantly to the cost of hospital care and to the length of hospital stays.

Although most patients with nosocomial pneumonia develop fever and leukocytosis, these findings are not uniform and are not a requisite for the presumptive diagnosis of nosocomial pneumonia.

The ATS subdivides nosocomial pneumonia into early onset (usually within the first 4 d of the hospitalization) and late onset (usually occurring after the fifth hospital day). Early-onset nosocomial pneumonia tends to carry a better prognosis, whereas late-onset nosocomial pneumonia tends to be associated with multidrug-resistant organisms, meaning that it is associated with higher mortality rates.

Pathophysiology

The development of nosocomial pneumonia represents an imbalance between normal host defenses and the ability of microorganisms to colonize and then invade the lower respiratory tract. The primary route through which organisms enter the lower airways is via aspiration of oropharyngeal secretions into the trachea. Hematogenous spread to the lungs is an alternative but uncommon route of infection.

Because aerobic gram-negative bacilli are the major pathogens associated with nosocomial pneumonia, the pathophysiology relates to the destructive effect of these organisms on invaded lung tissue. Aerobic gram-negative pathogens may be divided into two categories. The first category includes organisms that cause necrotizing pneumonia with rapid cavitation, microabscess formation, blood-vessel invasion, and hemorrhage (eg, *Pseudomonas aeruginosa*). The second category consists of all other nonnecrotizing gram-negative organisms responsible for nosocomial pneumonia.

Frequency

United States

Nosocomial pneumonia is the second-most-common nosocomial infection in the United States and is usually bacterial in nature. It is one of the most common diagnoses made in medical and surgical intensive care units (ICUs) and is common in patients undergoing mechanical ventilation. Nosocomial pneumonia also occurs in patients in the general hospital wards who are not receiving mechanical ventilation.

International

The international incidence and prevalence of nosocomial pneumonia is similar to that in the United States, with comparable rates of responsible microorganisms.

Mortality/Morbidity

Because patients in ICUs are already typically critically ill, the mortality and morbidity rates associated with nosocomial pneumonia in these patients are high. Intubation and ventilatory support bypass the normal host defense mechanisms, predisposing to infection.

Race

Nosocomial pneumonia has no racial predilection.

Sex

Nosocomial pneumonia has no sexual predilection.

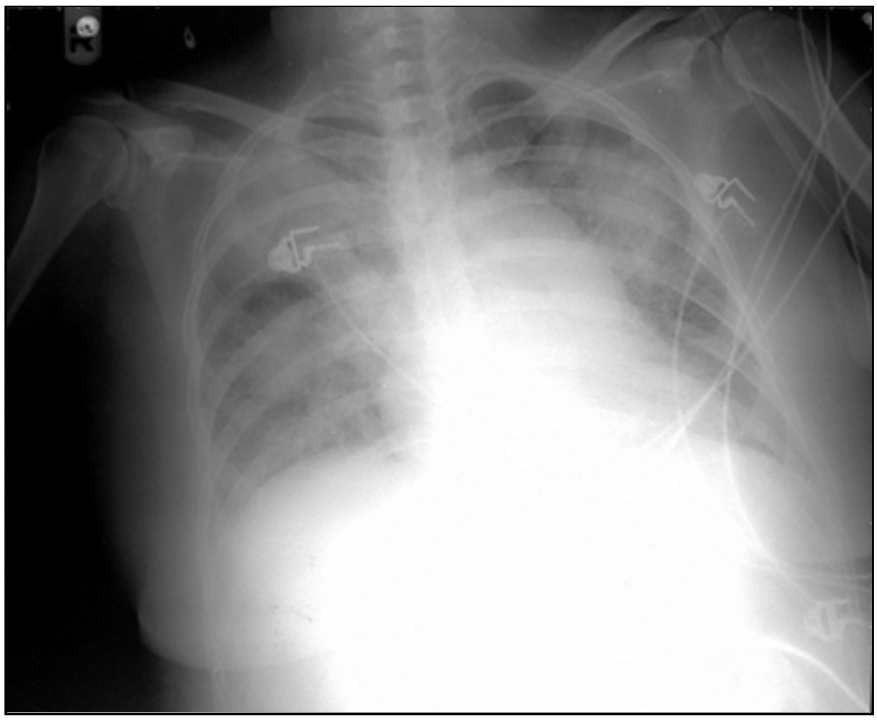
Age

Nosocomial pneumonia is most common in elderly patients; however, patients of any age may be affected.

Clinical

History

- Symptoms of nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP])
  - Respiratory tract symptoms, which may include an increase in respiratory rate, shortness of breath, and a productive cough
  - Fever (most cases)
- Clinical diagnosis
  - In most cases, the diagnosis of nosocomial pneumonia is clinical, supported by appropriate cultures, which may include semiquantitative cultures from bronchoalveolar lavage (BAL) samples.
  - The definitive diagnosis of nosocomial pneumonia rests on tissue biopsy, which is rarely performed; therefore, the clinician is forced to grapple with various nonspecific findings that can mimic nosocomial pneumonia. This situation is particularly true of chest radiographic findings of pulmonary infiltrates, which may be caused by numerous conditions common in the critical care setting. The most common causes of infiltrates in ventilated patients with fever and/or leukocytosis include the following conditions:
    - Congestive heart failure
    - Pulmonary embolus or infarction
    - Acute respiratory distress syndrome (ARDS)
    - Pulmonary drug reactions
    - Collagen vascular diseases (eg, systemic lupus erythematosus [SLE]), bronchiolitis obliterans-organizing pneumonia (BOOP), interstitial lung disease, bronchogenic carcinomas, metastatic carcinomas, radiographic artifacts (see Media file 1)



Typical chest radiograph of a patient with nosocomial pneumonia.

Physical

- Physical findings in nosocomial pneumonia relate to the pneumonia distribution in the chest. Physically, lobar lesions caused by nosocomial pneumonia mimic those caused by any other type of pneumonia (eg, rales in the location of the pneumonic process).
- In most cases, neither consolidation nor pleural effusions are features of nosocomial pneumonia. The presence of either should prompt consideration of an alternate diagnosis.
- The presumptive diagnosis of nosocomial pneumonia is clinical and is based on nonspecific findings; therefore, it is not necessarily precise. Most patients in the ICU who have fever and pulmonary infiltrates probably do not have nosocomial pneumonia; nonetheless, therapy should be based on a clinical diagnosis since tissue-based biopsy methods are not used in most patients.
- Several conditions mimic nosocomial pneumonia; therefore, incorporate the exclusion of these conditions as part of the clinical diagnosis.

Causes

Inhalation, aspiration, and hematogenous spread are the 3 main mechanisms by which bacteria reach the lungs.

Factors that predispose to infection include the following:

- Primary inhalation pneumonia develops when these organisms bypass normal respiratory defense mechanisms or when the patient inhales aerobic gram-negative organisms that colonize the upper respiratory tract or respiratory support equipment.
- Aspiration pneumonia is due to aspiration of colonized upper respiratory tract secretions.

- The stomach appears to be an important reservoir of gram-negative bacilli that can ascend and colonize the respiratory tract. A prospective observational study found that patients who used acid-suppressive medications were more likely to develop hospital-acquired pneumonia than were patients who did not (5% vs 2%). Further evaluation by drug class showed that the risk for pneumonia was significantly increased with proton pump inhibitors, but not with histamine 2–blocking agents.[1]
- Hematogenously acquired infections originate from a distant source and reach the lungs via the bloodstream. In bacteremic nosocomial pneumonia, blood culture results are frequently positive if obtained early in the disease process and if the patient is not already receiving antimicrobial therapy.

Microbiology of Nosocomial Pneumonia

Common causes of nosocomial pneumonia

Common bacteria involved in nosocomial pneumonia include the following:

- *P aeruginosa*
- *Klebsiella* species
- *Escherichia coli*
- *Acinetobacter* species (*Acinetobacter* species commonly colonize the respiratory tract secretions in patients in the ICU. Care must be exercised in interpretation of culture data.)
- *Staphylococcus aureus*, especially methicillin-resistant *S aureus* (MRSA)
- *Streptococcus pneumoniae* (should be considered in early-onset HAP; causes up to 9% of pneumonias in elderly patients in nursing homes)
- *Haemophilus influenzae* (should be considered in early-onset HAP)

Nosocomial pneumonia is mistakenly diagnosed in many cases; therefore, the differential diagnoses are important.

Less-common pathogens associated with nosocomial pneumonia

The following are less-common pathogens implicated in nosocomial pneumonia:

- *Serratia* species
- *Legionella* species (*Legionella* nosocomial pneumonia occurs only in outbreaks or clusters.)
- Influenza A virus
- Respiratory syncytial virus (RSV)
- Parainfluenza virus
- Adenovirus

Influenza A virus, RSV, parainfluenza virus, and adenovirus, may cause HAP in the right clinical setting.

Extremely rare causes of nosocomial pneumonia

The following are rarely isolated in patients with nosocomial pneumonia:

- *Enterobacter* species
- *Stenotrophomonas maltophilia* (formerly *Pseudomonas maltophilia*)
- *Burkholderia cepacia* (formerly *Pseudomonas cepacia*)
- *Candida* species (*Candida* species are an uncommon cause of HAP, and cultures positive for these organisms more often reflect colonization.)
- Oropharyngeal anaerobes (non– *Bacteroides fragilis*)

Although these organisms may be very uncommon causes of nosocomial pneumonia, they have been recovered in patients with ventilator-associated pneumonia (VAP). The recovery from respiratory secretions of an organism that is typically pathogenic does not prove that it is pathogenic or the cause of nosocomial pneumonia. Anaerobic organisms are not typically isolated in nosocomial pneumonia.

Multiple organisms as a cause of nosocomial pneumonia

Multiple pathogens are proof of lower airway colonization obtained by nontissue biopsy culture methods.

Differential Diagnoses

Acute Respiratory Distress Syndrome
Pulmonary Edema, Cardiogenic
Pulmonary Embolism

Workup

Laboratory Studies

All patients with presumed nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP]) should undergo testing to rule out conditions that mimic nosocomial pneumonia. The presumptive diagnosis of nosocomial pneumonia is difficult because the diagnosis does not depend on the presence of fever, and leukocytosis is unhelpful. A summary of management strategies is available through a practice guideline provided by the ATS.[2]

- White blood cell count
  - A white blood cell (WBC) count is usually suggested but does not yield a specific finding.
  - The WBC count may be normal or elevated in cases of nosocomial pneumonia or conditions that mimic nosocomial pneumonia. A left shift reflects the stress that the patient is being subjected to and neither rules out nor confirms infection. The degree of left shift indicates the degree of stress in the host.
  - Neither leukocytosis nor a normal WBC count favors the diagnosis of nosocomial pneumonia over the diseases that mimic nosocomial pneumonia, as these can produce a similar elevation.
- Obtain blood cultures as early as possible to retrospectively diagnose infection with hematogenous pathogens.

Imaging Studies

- Obtain serial chest radiographs to assist in evaluating the progress of the pneumonia and the efficacy of appropriate antimicrobial therapy.
- Radiographs may also be useful in distinguishing various mimics from actual nosocomial pneumonia. In

these patients, CT scanning or spiral CT scanning may be useful.

Other Tests

- ECGs and ventilation-perfusion scans should eliminate pneumonia mimics. ECGs, cardiac enzymes, and Swan-Ganz readings may rule out left ventricular failure caused by exacerbation of heart failure or new myocardial infarction.
- Obtain other tests that are related to the possible underlying causes of the pulmonary infiltrates; for example, if lupus pneumonitis is suspected, ask the patient about a history of SLE pneumonitis. Afterward, serologic tests should be performed to assess for SLE.
- Tests such as arterial blood gas (ABG) studies are merely used to assess the degree of severity of lung dysfunction but are not useful in diagnosing nosocomial pneumonia. Obtain ABGs to help diagnose a diffusion defect related to interstitial lung diseases.

Procedures

- Bronchoscopic techniques
  - These techniques yield variable sensitivities and specificities, although there are accepted criteria for semiquantitative cultures to improve the diagnostic reliability of bronchoscopically derived cultures.
  - A bronchoscopic bacteriologic strategy has been shown to reduce the short-term mortality risk in one study.

Histologic Findings

Histologic study of lung tissue reveals either necrotizing pneumonia or nonnecrotizing pneumonia, depending on the pathogen. *P aeruginosa* produces a necrotizing pneumonia with vessel invasion, local hemorrhage, and microabscess formation. Other aerobic gram-negative bacilli produce a polymorphonuclear response at the site of invasion, but microabscess formation and vessel invasion are absent.

Treatment

Medical Care

Patients with nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP]) usually require ventilatory support at some point and usually need supplemental oxygen therapy.

Before empiric antimicrobial therapy is initiated, an attempt should be made to rule out mimics of nosocomial pneumonia. If mimics of nosocomial pneumonia can be excluded with a reasonable degree of certainty, then empiric therapy for nosocomial pneumonia is appropriate.

The precise pathogen that causes a given case of nosocomial pneumonia is usually unknown. Therefore, empiric antimicrobial therapy is the only practical approach. Delaying therapy until the pathogen is identified is not recommended. For empiric coverage of nosocomial pneumonia, monotherapy is as effective as combination therapy for early nosocomial pneumonia.

For proven pseudomonal nosocomial pneumonia, double-drug coverage with a high degree of antipseudomonal activity and a low resistance potential should be used. Optimal combinations include meropenem or doripenem plus either levofloxacin or aztreonam. Alternately, antipseudomonal penicillin (eg, piperacillin) in combination with levofloxacin, meropenem, aminoglycoside, or aztreonam may provide equal efficacy.

Principles of appropriate empiric antibiotic coverage in nosocomial pneumonias

Direct empiric coverage against common nosocomial pathogens *P aeruginosa, Klebsiella* species, *E coli, and* MRSA. Coverage against *P aeruginosa* also covers other nosocomial pneumonia pathogens.

Enterobacter species, *S maltophilia, and Burkholderia cepacia: Enterobacter* species usually do not cause nosocomial pneumonia. *S maltophilia* and *B cepacia* are common colonizers of respiratory secretions but rarely, if ever, cause nosocomial pneumonia in most hosts; however, they are potential pathogens in patients with bronchiectasis or cystic fibrosis.

Oropharyngeal anaerobes are unimportant from a therapeutic standpoint, as they are not typically isolated in nosocomial pneumonia.

Empiric monotherapy versus combination therapy

The optimal empiric monotherapy for nosocomial pneumonia consists of ceftriaxone, ertapenem, levofloxacin, or moxifloxacin. Monotherapy may be acceptable in patients with early-onset HAP. Avoid monotherapy with ciprofloxacin, ceftazidime, or imipenem, as they are likely to induce resistance potential. Late-onset HAP, ventilator-associated pneumonia (VAP), or HCAP requires combination therapy using an antipseudomonal cephalosporin, beta-lactam, or carbapenem plus an antipseudomonal fluoroquinolone or aminoglycoside plus an agent such as linezolid or vancomycin to cover MRSA.

Optimal combination regimens for proven *P aeruginosa* nosocomial pneumonia include (1) piperacillin/tazobactam plus amikacin or (2) meropenem plus levofloxacin, aztreonam, or amikacin. Avoid using ciprofloxacin, ceftazidime, gentamicin, or imipenem in combination regimens, as combination therapy does not eliminate the resistance potential of these antibiotics. When selecting an aminoglycoside for a combination therapy regimen, amikacin once daily is preferred to gentamicin or tobramycin to avoid resistance problems. When selecting a quinolone in a combination therapy regimen, use levofloxacin, which has very good anti–*P aeruginosa* activity (equal or better than ciprofloxacin at a dose of 750 mg).

Consultations

- Consult an infectious disease specialist to assess the microbiology of the specimens obtained from the patient, to rule out the mimics of nosocomial pneumonia, and to administer empiric or specific empirical antimicrobial therapy.
- Consult a pulmonologist to help with mechanical ventilation (often required in patients with nosocomial pneumonia).
- Other consultations include the following:
  - Rheumatologist, if the patient appears to have lupus or SLE pneumonitis
  - Cardiologist, if the patient has heart failure
  - Oncologist for possible pulmonary infiltrates caused by a lymphangitic spread of a malignancy

Diet

- Most patients with nosocomial pneumonia are intubated and are instructed to receive nothing by mouth (NPO).

Activity

- Most patients with nosocomial pneumonia are intubated and are limited to bed rest.

Medication

Ordinarily, nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP]) is treated for 14 days. If the patient indeed has nosocomial pneumonia and the appropriate antimicrobial therapy is administered, chest radiography shows significant improvement in the pulmonary infiltrate during the 2 weeks of antimicrobial therapy. Pulmonary infiltrates that are unchanged after a 2-week course of therapy suggest that the infiltrates may not be infectious in origin. Start a diagnostic workup to consider other infectious diseases that do not respond to antibiotics (eg, herpesvirus type 1 [HSV-1] pneumonitis) or noninfectious diseases (eg, bronchogenic carcinomas).

Antibiotics

Empiric antimicrobial therapy must be comprehensive and should cover all likely pathogens in the context of the clinical setting.

Cefepime (Maxipime)

A fourth-generation cephalosporin with good gram-negative coverage similar to ceftazidime; however, better gram-positive coverage than ceftazidime is equivalent in its coverage of *P aeruginosa*. This drug may be more active than ceftazidime against *Enterobacter* species because of its enhanced stability against beta-lactamases.

Dosing

Adult

2 g IV q12h

Pediatric

50 mg/kg IV q8h

Interactions

Probenecid at a high dose decreases cefepime clearance; vancomycin, polymyxin B, colistin, loop diuretics, and aminoglycosides increase the risk of nephrotoxicity

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Adjust dose in patients with severe renal insufficiency

Meropenem (Merrem)

A carbapenem, not a beta-lactam antibiotic. Bactericidal broad-spectrum carbapenem antibiotic that inhibits cell wall synthesis. Effective against most gram-positive and gram-negative bacteria. Has slightly increased activity against gram-negative bacteria and a slightly decreased activity against staphylococci and streptococci when compared to imipenem.

Dosing

Adult

1 g IV q8h (normal renal function)

Pediatric

<10 years: Not established
>10 years: Administer as in adults

Interactions

Probenecid may inhibit renal excretion and increase meropenem levels

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Pseudomembranous colitis and thrombocytopenia may occur, requiring immediate discontinuation of medication

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Piperacillin (Pipracil)

Antipseudomonal penicillin. Acts on bacterial cell walls. Greatest degree of antipseudomonal activity among the antipseudomonal penicillins. Inhibits biosynthesis of cell wall mucopeptides and stage of active multiplication; has antipseudomonal activity. Used in combination with other antibiotics.

Dosing

Adult

4 g IV q8h (normal renal function)

Pediatric

<10 years: Not established
>10 years: Administer as in adults

Interactions

Tetracyclines may decrease effects; piperacillin at high concentrations may physically inactivate aminoglycosides; probenecid may increase levels of piperacillin; coadministration with aminoglycosides has synergistic effects

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

May interfere with platelet function in patients requiring surgery; caution in renal impairment and in history of seizures

Aztreonam (Azactam)

A monobactam, not a beta-lactam antibiotic, inhibits cell wall synthesis during bacterial growth. Active against gram-negative bacilli.

Dosing

Adult

2 g IV q8h (normal renal function)

Pediatric

<10 years: Not established
>10 years: Administer as in adults

Interactions

Tetracyclines may reduce effects of this medication

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Adjust dose in renal insufficiency

Amikacin (Amikin)

Used for gram-negative bacterial coverage of infections resistant to gentamicin and tobramycin. Effective against *P aeruginosa*. Irreversibly binds to 30S subunit of bacterial ribosomes, blocks recognition step in protein synthesis, and causes growth inhibition. Use patient's ideal body weight for dosage calculation.

Dosing

Adult

15 mg/kg/d IV/IM divided bid; not to exceed 1.5 g/d regardless of higher body weight

Pediatric

Administer as in adults

Interactions

Coadministration with other aminoglycosides, penicillins, cephalosporins, and amphotericin B increases nephrotoxicity; enhances effects of neuromuscular blocking agents; causes respiratory depression; irreversible hearing loss may occur with coadministration of loop diuretics

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Not intended for long-term therapy; caution in patients with renal failure (not on dialysis), hypocalcemia, myasthenia gravis, and conditions that depress neuromuscular transmission

Levofloxacin (Levaquin)

Second-generation quinolone. Acts by interfering with DNA gyrase in bacterial cells. This is a bactericidal and is highly active against gram-negative and gram-positive organisms including *P aeruginosa*. For pseudomonal infections and infections caused by multidrug-resistant gram-negative organisms.

Dosing

Adult

750 mg PO/IV q24h (normal renal function)

Pediatric

<18 years: Not recommended
>18 years: Administer as in adults

Interactions

Antacids, iron salts, and zinc salts may reduce serum levels; administer antacids 2-4 h before or after taking fluoroquinolones; cimetidine may interfere with metabolism of fluoroquinolones; levofloxacin reduces therapeutic effects of phenytoin; probenecid may increase levofloxacin serum concentrations; may increase toxicity of theophylline, caffeine, cyclosporine, and digoxin (monitor digoxin levels); may increase effects of anticoagulants (monitor PT)

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

In prolonged therapy, perform periodic evaluations of organ system functions (eg, renal, hepatic, hematopoietic); adjust dose in renal function impairment; superinfections may occur with prolonged or repeated antibiotic therapy

Piperacillin and tazobactam sodium (Zosyn)

Antipseudomonal penicillin plus beta-lactamase inhibitor. Inhibits biosynthesis of cell wall mucopeptide and is

effective during stage of active multiplication.

Dosing

Adult

4.5 g (piperacillin 4 g and tazobactam 0.5 g) IV q6h

Pediatric

<12 years: Not established
>12 years: Administer as in adults

Interactions

Tetracyclines may decrease effects of piperacillin; high concentrations of piperacillin may physically inactivate aminoglycosides if administered in same IV line; effects when administered concurrently with aminoglycosides are synergistic; probenecid may increase penicillin levels; high-dose parenteral penicillins may result in increased risk of bleeding

Contraindications

Documented hypersensitivity; severe pneumonia, bacteremia, pericarditis, emphysema, meningitis and purulent or septic arthritis should not be treated with an oral penicillin during the acute stage

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Perform CBC count prior to initiation of therapy and at least weekly during therapy; monitor for liver function abnormalities by measuring AST and ALT levels during therapy; exercise caution in patients with hepatic insufficiencies; perform urinalysis and BUN and creatinine determinations during therapy and adjust dose if values become elevated; monitor blood levels to avoid possible neurotoxic reactions

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Doripenem (Doribax)

Carbapenem antibiotic. Elicits activity against a wide range of gram-positive and gram-negative bacteria. Indicated as a single agent for complicated intra-abdominal infections caused by susceptible strains of *E coli, Klebsiella pneumoniae, P aeruginosa, Bacteroides caccae, B fragilis, Bacteroides thetaiotaomicron, Bacteroides uniformis, Bacteroides vulgatus, Streptococcus intermedius, Streptococcus constellatus,* and *Peptostreptococcus micros.*

Dosing

Adult

500 mg IV q8h infused over 1 h
CrCl 30-49: 250 mg IV q8h
CrCl 11-29: 250 mg IV q12h

Pediatric

<18 years: Not established
>18 years: Administer as in adults

Interactions

Carbapenems may decrease valproic acid serum concentration, causing increased seizure risk; probenecid reduces renal clearance of doripenem, resulting in increased doripenem concentration; does not inhibit or induce major CYP450 enzymes

Contraindications

Documented hypersensitivity to doripenem or other carbapenems or demonstrated anaphylactic reactions to beta-lactams

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Clostridium difficile –associated diarrhea has been reported with nearly all antibacterial agents and must be considered if patient presents with diarrhea; common adverse effects (ie, >5%) include headache, nausea, diarrhea, rash, and phlebitis; decrease dose with renal insufficiency

Follow-up

Further Inpatient Care

- Patients with nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP]) should be transferred to the ICU if they are on the general medical wards and cannot be maintained without ventilatory support.
- Patients in the ICU may require ventilatory support, depending on their respiratory status.

Deterrence/Prevention

- Beds that permit some degree of patient turning may decrease the risk of nosocomial pneumonia in at-risk patients.

Complications

- The list of differential diagnoses represents the main clinical problem in nosocomial pneumonia. Many conditions mimic the presentation of nosocomial pneumonia. For example, any disorder that results in leukocytosis with variable degrees of left shift may be included in the differential diagnoses. An inflammatory or infectious process can cause fever; therefore, do not regard this symptom as an indication of an infectious disease process. Many conditions other than nosocomial pneumonia can cause pulmonary infiltrates. Consider all of these differential diagnoses carefully before settling on a diagnosis and embarking on a course of antimicrobial therapy.
- Failure to successfully wean the patient from the respirator (possibly because of lack of cardiopulmonary function or a superimposed process [eg, HSV-1 pneumonitis]) is a common problem following intubation for nosocomial pneumonia.
- HSV-1 pneumonitis develops in intubated patients who have unchanging or persistent pulmonary infiltrates after 2 weeks of antimicrobial therapy. These patients usually have low-grade fevers with variable degrees of leukocytosis. Demonstrating HSV-1 in samples of respiratory secretions may establish the diagnosis.
- Start treatment with acyclovir in patients diagnosed with HSV-1 infection; acyclovir decreases hypoxemia

and subsequently permits weaning of the patient from the respirator.

Prognosis

- The prognosis in patients with nosocomial pneumonia depends on preexisting underlying cardiopulmonary function and host defenses.

Miscellaneous

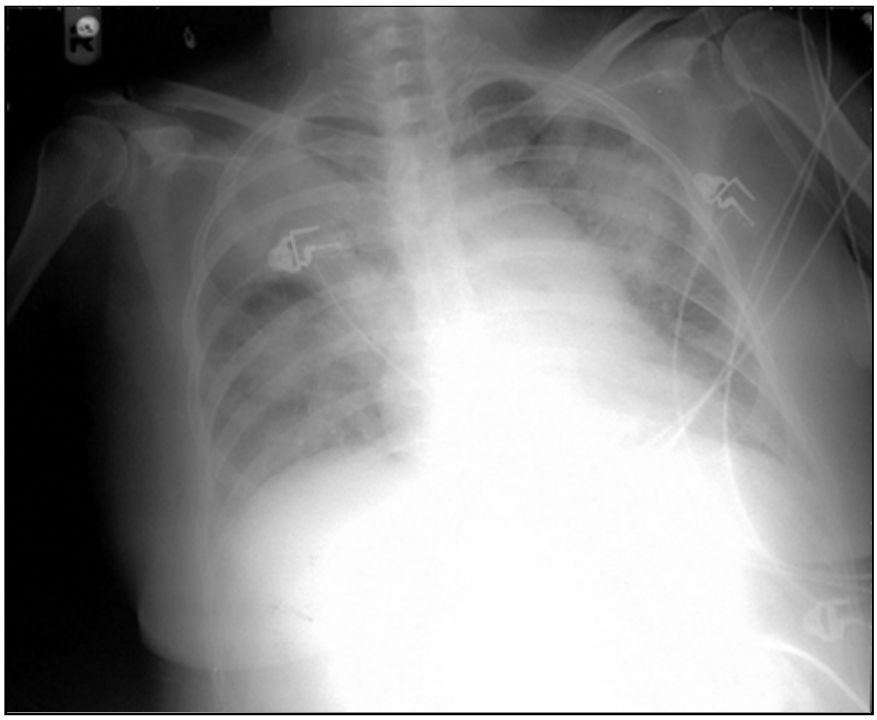
Medicolegal Pitfalls

- Failure to direct empiric monotherapy against *P aeruginosa,* ensuring coverage against all other bacteriologic causes of nosocomial pneumonia (NP; also known as hospital-acquired pneumonia [HAP] or health care–associated pneumonia [HCAP])
- Failure to consider the numerous conditions that mimic nosocomial pneumonia, many of which are treatable and reversible disorders
- Failure to consider the most common conditions that mimic nosocomial pneumonia, which include pulmonary hemorrhage, pulmonary embolus, and congestive heart failure
- Failure to suspect ARDS, which is usually readily diagnosable according to the microatelectatic changes on the chest radiograph and the progressive and severe hypoxemia indicated by the ABG levels (Little or no fever may accompany these symptoms.)

Special Concerns

- Compromised cardiac and lung function may further decrease the cardiopulmonary reserve of pneumonia, accounting for the high mortality and morbidity rates associated with nosocomial pneumonia.
- Barotrauma may decrease an already compromised lung function and alter chest radiographic appearances.

Multimedia



Media file 1: Typical chest radiograph of a patient with nosocomial pneumonia.

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Keywords

nosocomial pneumonia, NP, hospital-acquired pneumonia, HAP, healthcare-associated pneumonia, health care–associated pneumonia, HCAP, ventilator-associated pneumonia, VAP

Contributor Information and Disclosures

Author

Burke A Cunha, MD, Professor of Medicine, State University of New York School of Medicine at Stony Brook; Chief, Infectious Disease Division, Winthrop-University Hospital
Burke A Cunha, MD is a member of the following medical societies: American College of Chest Physicians, American College of Physicians, and Infectious Diseases Society of America
Disclosure: Nothing to disclose.

Medical Editor

Wesley W Emmons, MD, FACP, Assistant Professor, Department of Medicine, Thomas Jefferson University; Consulting Staff, Infectious Diseases Section, Department of Internal Medicine, Christiana Care, Newark, DE
Wesley W Emmons, MD, FACP is a member of the following medical societies: American College of Physicians, American Medical Association, American Society of Tropical Medicine and Hygiene, Infectious Diseases Society of America, and International AIDS Society
Disclosure: Nothing to disclose.

Pharmacy Editor

Francisco Talavera, PharmD, PhD, Senior Pharmacy Editor, eMedicine
Disclosure: Nothing to disclose.

Managing Editor

Ronald A Greenfield, MD, Professor, Department of Internal Medicine, Section of Infectious Diseases, University of Oklahoma College of Medicine
Ronald A Greenfield, MD is a member of the following medical societies: American College of Physicians, American Federation for Medical Research, American Society for Microbiology, Central Society for Clinical Research, Infectious Diseases Society of America, Medical Mycology Society of the Americas, Phi Beta Kappa, Southern Society for Clinical Investigation, and Southwestern Association of Clinical Microbiology
Disclosure: Pfizer Honoraria Speaking and teaching; Gilead Honoraria Speaking and teaching; Ortho McNeil Honoraria Speaking and teaching; Wyeth Honoraria Speaking and teaching; Abbott Honoraria Speaking and teaching; Astellas Honoraria Speaking and teaching; Cubist Speaking and teaching

CME Editor

Eleftherios Mylonakis, MD, Clinical and Research Fellow, Department of Internal Medicine, Division of Infectious Diseases, Massachusetts General Hospital
Eleftherios Mylonakis, MD is a member of the following medical societies: American Association for the Advancement of Science, American College of Physicians, American Society for Microbiology, and Infectious Diseases Society of America
Disclosure: Nothing to disclose.

Chief Editor

Michael Stuart Bronze, MD, Professor, Stewart G Wolf Chair in Internal Medicine, Department of Medicine, University of Oklahoma Health Science Center
Michael Stuart Bronze, MD is a member of the following medical societies: Alpha Omega Alpha, American College of Physician Executives, American College of Physicians, American College of Physicians-American Society of Internal Medicine, American Federation for Clinical Research, American Medical Association, American Society for Microbiology, Association of Professors of Medicine, Association of Program Directors in Internal Medicine, Infectious Diseases Society of America, Oklahoma State Medical Association, and Southern Society for Clinical Investigation
Disclosure: Nothing to disclose.

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