Acute Exacerbation of Chronic Bronchitis: A Primary Care Consensus Guideline

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Objective: To develop consensus on appropriate treatment for acute exacerbation of chronic bronchitis (AECB).

Characteristics and Etiology: Patients with chronic bronchitis have an irreversible reduction in maximal airflow velocity and a productive cough on most days of the month for 3 months over 2 consecutive years. An AECB is characterized by a period of unstable lung function with worsening airflow and other symptoms. Most (80%) cases of AECB are due to infection, with half due to aerobic bacteria. The remaining 20% are due to noninfectious causes such as environmental factors or medication nonadherence.

Management: Supportive care should be provided to all patients, which might include removal of irritants, use of a bronchodilator, oxygen, hydration, use of a systemic corticosteroid, and chest physical therapy. Antibacterial treatment should be reserved for patients with at least 1 key symptom (ie, increased dyspnea, sputum production, sputum purulence) and 1 risk factor (ie, age ≥ 65 years, forced expiratory volume in 1 second <50% of the predicted value, ≥4 AECBs in 12 months, 1 or more comorbidities). A newer macrolide, extended-spectrum cephalosporin, or doxycycline is appropriate for an exacerbation of moderate severity, and high-dose amoxicillin/clavulanate or a respiratory fluoroquinolone should be used for a severe exacerbation. There has been increasing antibacterial resistance by the 3 most prevalent pathogens (*Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*).

Conclusion: Although all AECB patients should receive supportive care, only patients with at least 1 key symptom and 1 risk factor should receive antibiotic therapy.

(Am J Manag Care. 2004;10:689-696)

Chronic bronchitis is a subset of disease within the broader category of chronic obstructive pulmonary disease (COPD), which is characterized by a chronic, predominantly irreversible reduction in maximal airflow velocity. Chronic bronchitis, emphysema, asthma, and to a lesser extent bronchiectasis comprise the majority of COPD (**Figure 1**). Chronic bronchitis is a disease process identified clinically as the presence of a productive cough on most days of the month for 3 months over 2 consecutive years.[1] It occurs in the absence of other causes of chronic cough such as tuberculosis or lung cancer. An acute exacerbation of chronic bronchitis (AECB) is a distinct event superimposed on chronic bronchitis and is characterized by a period of unstable lung function with worsening airflow and other symptoms. The average number of episodes of AECB per year is reported to range from 1.5 to 3.[2-4]

IMPACT OF ACUTE EXACERBATIONS OF CHRONIC BRONCHITIS

The overall rate of emergency department visits for chronic bronchitis increased 28% between 1992 and 2000. The rate of visits rose more significantly for African Americans than Caucasians. The rate increased in all age groups, particularly in persons aged 55 to 64 years; in fact, the rate in this group now approaches the rate in persons aged 65 years or older (**Figure 2**).[5] Needless to say, the health and socioeconomic consequences are enormous. A retrospective analysis of Medicare and other databases involving more than 280 000 patients with AECB showed that the total cost of treatment in 1994 was approximately $1.6 billion.[6] Outpatient care accounted for only $40 million (2.5% of the total cost) or approximately $70 per visit. This clearly demonstrates that hospitalization due to AECB accounts for the vast majority of total expenditures. A more recent report found the cost of inpatient hospitalization for AECB ranged from $6285 to $6625.[7]

The impact on families and informal caregivers also is substantial because they provide an average of 5.1 hours per week of informal care to patients with emphy-

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This research was supported by an educational grant from Ortho-McNeil Pharmaceutical, Inc.

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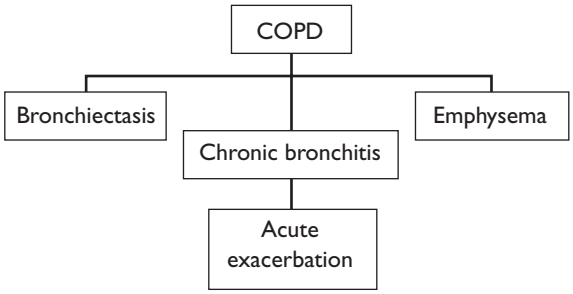
Figure 1. Subsets of Chronic Obstructive Pulmonary Disease (COPD)



sema or chronic bronchitis.[8] Undoubtedly, the impact is even greater during the period when a patient with chronic bronchitis has an episode of AECB.

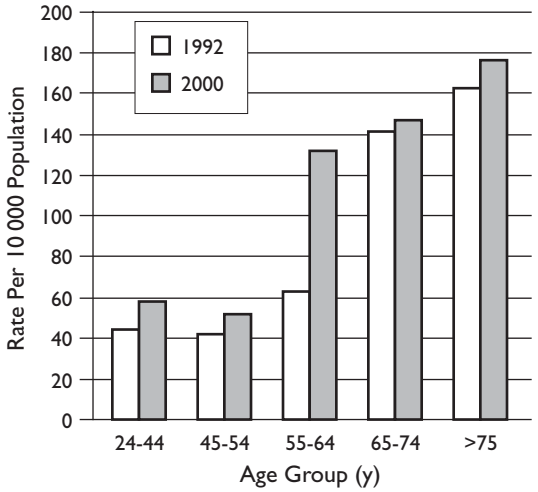
CLINICAL ASSESSMENT

The purpose of the initial clinical assessment of patients with AECB is twofold. First, it should serve to determine whether the worsening respiratory status is due to a concomitant disease or a trigger for an acute exacerbation. Second, it should determine the severity of illness so as to guide management and predict prognosis.

The diagnosis of AECB generally is made on clinical grounds. Many factors must be assessed during the history, physical examination, and workup (**Table 1**).[9-11] Although acute exacerbations generally result from bacterial or viral infections, 20% of cases have a noninfectious cause.[12] Exposure to allergens, pollutants, or cigarette smoke must be considered, and the importance of that exposure must be assessed.

A change from baseline in 1 or more chronic symptoms generally indicates worsening disease. Such symptoms include shortness of breath, increased sputum production, increased sputum purulence, cough, and increased sputum tenacity. The first 3 of these symptoms—shortness of breath, increased sputum production, and increased sputum purulence—are particularly helpful to determine the severity of the exacerbation.[4,13] Patients with only 1 of these 3 symptoms generally are considered to have a mild acute exacerbation; those with 2 of the 3 symptoms, a moderate acute exacerbation; and those with all 3 symptoms, a severe acute exacerbation (**Table 2**). Clinically, increased dyspnea, cold symptoms, and sore throat are associated with a viral exacerbation, whereas an exacerbation characterized by increased sputum production or purulence, and associated with neutrophilic inflammation, is likely to be bacterial in nature.[3,14,15] In fact, evolving evidence indicates that some markers of inflammation, such as interleukin-6, interleukin-8, tumor necrosis factor-alpha, neutrophil elastase, and serum fibrinogen, may be useful to distinguish bacterial from nonbacterial AECB, as well as the bacterial etiology.[14] Although the measurement of these inflammatory markers is limited to the research setting at present, their use in clinical practice is possible in the near future.

Figure 2. Rates of Emergency Department Visits With Chronic Obstructive Pullmonary Disease as the First-Listed Diagnosis, by Age



Although utilization of the 3 symptoms just discussed is helpful to assess the severity of the acute exacerbation, false positives and false negatives are frequent. Unfortunately, the diagnostic usefulness of a culture remains contentious because bacterial pathogens can be isolated from the sputum of patients with stable chronic bronchitis (ie, bacterial colonization) as frequently as they can from the sputum of patients with AECB.[9,14,16] Interestingly, however, it has been observed that a new strain of a bacterial pathogen was isolated twice as frequently during AECB as it was during stable chronic bronchitis.[17] A sputum culture may, however, be useful in certain situations such as recurrent AECB, an inadequate response to therapy, and before starting treatment with prophylactic antibiotics.[12]

A chest radiograph is not used to diagnose AECB, but it may be helpful in patients who have an atypical presentation and in whom community-acquired pneumonia is suspected.[9] In addition, a chest radiograph is helpful to identify comorbidities that may contribute to the acute exacerbation. The presence of pulmonary

edema or pulmonary infiltrate on chest radiograph is more useful than history and clinical signs and symptoms to identify patients with congestive heart failure or pneumonia, respectively.[18]

Assessment of oxygen saturation is important to guide therapy. Indirect evidence from several studies indicates that arterial blood gas analysis is helpful to gauge the severity of an exacerbation and to identify those patients in need of oxygen therapy, as well as those who might require mechanical ventilation.[16] Although commonly used in the assessment of AECB, the benefit of pulse oximetry has not been investigated in a clinical trial.[19]

Although the role of spirometry in diagnosis of AECB is less clear than it is in diagnosis of COPD,[16,19] evidence from 3 trials show that measurement of lung function using spirometry is valuable to assess the degree of airway obstruction.[20-22] The forced expiratory volume in 1 second (FEV_1) is correlated with the partial pressure of carbon dioxide ($PaCO_2$) and pH, but not with the partial pressure of oxygen (PaO_2). The FEV_1 also is correlated with the relapse rate. Spirometry is available in emergency departments and increasingly in the primary care setting. The computer-assisted devices now available are relatively simple to use with appropriate training.[23] Although some patients in respiratory distress are not able to perform full spirometry to assess FEV_1, the use of a peak flow meter is not appropriate because the peak expiratory flow rate is not sufficiently well correlated with lung function to substitute for FEV_1.[18] It should be noted, however, that serial measurement of the peak expiratory flow rate with a peak flow meter might be clinically useful.[19] Similarly, baseline spirometry should be undertaken in all smokers who are without symptoms at present. Not only will this help to uncover existing lung dysfunction, but it also can be helpful for comparative purposes during an acute exacerbation.

ETIOLOGY OF ACUTE EXACERBATION OF CHRONIC BRONCHITIS

The infectious and noninfectious causes of AECB have been historically difficult to quantify because of difficulty in isolating organisms, in differentiating between pathogens and colonized organisms, and in defining patients with AECB in clinical trials, to name a few reasons. A review by Sethi of the relevant literature led him to conclude that 80% of AECB cases are infectious in nature, and noninfectious causes such as environmental factors or triggers and medication nonadherence comprise the remainder.[12]

Table 1. Key Assessment Factors

- Age
- Triggers
- Comorbid diseases
- Response to previous medical therapy
- Overall pulmonary function
- Oxygenation
- Character and severity of previous exacerbations
- Bacterial colonization status
- Previous need for mechanical ventilation
- Local antimicrobial susceptibility pattern

In cases of AECB due to infection, 3 classes of pathogens have been found: aerobic gram-positive and gram-negative bacteria, respiratory viruses, and atypical bacteria (**Figure 3**).[12] Although the review by Sethi was not intended to rigorously quantify the incidence of specific pathogens, he observed that aerobic bacteria were found in half of patients with AECB and viruses in one third. The predominant aerobic bacteria are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.[12] *Pseudomonas aeruginosa* and other gram-negative bacilli also are seen and appear to be more common in patients who have a severe acute exacerbation with an FEV_1 of 35% or less of the predicted value.[24] Infection due to multiple pathogens occurs in a small percentage of all patients with AECB and is more common in patients with a severe exacerbation.[12]

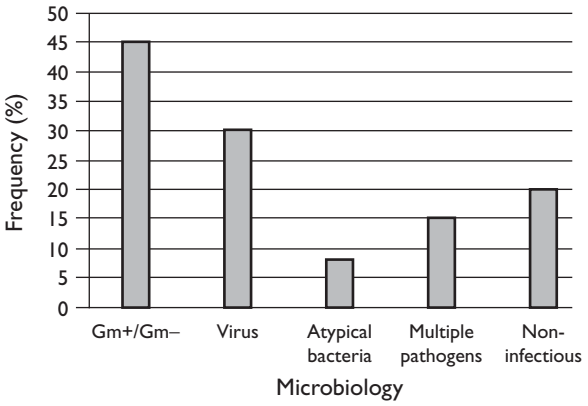
Viral infection is commonly associated with AECB. In 1 study, 64% of exacerbations were associated with a cold that occurred within the previous 18 days.[3] This study showed that patients with a cold experienced increased dyspnea, had a higher total symptom count at presentation, and had a median symptom recovery period of 13 days. The pattern of viral pathogens is variable. One study found that a rhinovirus was identified in 58% of exacerbations, and a respiratory syncytial virus,

Table 2. Symptom-related Severity of Acute Exacerbation of Chronic Bronchitis

No. of Key Symptoms*	Severity
1	Mild
2	Moderate
3	Severe

*Key symptoms are increased dyspnea, increased sputum production, and increased sputum purulence.

Figure 3. Etiology of Acute Exacerbation of Chronic Bronchitis



coronavirus, or influenza A virus was found in 29%, 11%, and 9%, respectively.[12] A review of 3 longitudinal studies, on the other hand, showed that influenza was the viral pathogen most frequently observed and was found in one third of patients, while parainfluenza was isolated in one quarter of patients.[12] Nonetheless, these findings support the importance of preventing colds (primarily by hand washing and avoiding exposure to those with a cold) and other viral infections, and stress the value of yearly influenza immunization.

Fewer than 10% of acute exacerbations are due to an atypical bacterium. The most common atypical bacterium is *Chlamydia pneumoniae*, whereas *Mycoplasma pneumoniae* and *Legionella pneumophila* are seen less frequently.[12]

PATTERNS OF ANTIBACTERIAL RESISTANCE

The susceptibilities of *S pneumoniae*, *H influenzae*, and *M catarrhalis* to antibacterial agents have changed dramatically over the past decade. The Tracking Resistance in the United States Today (TRUST) study has tracked resistance at national and regional levels in the United States since 1996. During the 2001-2002 respiratory season (TRUST 6), approximately 10 000 isolates were collected from adult and pediatric inpatients and outpatients at 239 institutions across the 9 US Bureau of the Census regions.[25] These data indicate that the susceptibility patterns continue to change (**Table 3**). Resistance of *S pneumoniae* to penicillin, azithromycin (and other macrolides), trimethoprim/sulfamethoxazole, and cefuroxime continues to be high. On the other hand, resistance to amoxicillin/clavulanate, ceftriaxone, levofloxacin, and vancomycin remains low. For *H influenzae*, resistance to ampicillin and trimethoprim/sulfamethoxazole is high, although it remains low for the other agents tested. β-Lactamase production by *M catarrhalis* continues to significantly increase the minimum inhibitory concentration for 90% of strains for β-lactam antibiotics, particularly ampicillin and cefuroxime. The correlation of in vitro resistance and clinical efficacy remains unclear.

The TRUST 6 data also show considerable geographic variation. Generally, the susceptibilities of *S pneumoniae* are lowest in the South Atlantic states and highest in New England and the Pacific states. More than 88% of *S pneumoniae* strains remain susceptible in the New England and Pacific states, but fewer than 80% are susceptible in the East South-Central and South Atlantic states. Although these data provide valuable information regarding general trends, knowledge of local susceptibility patterns is critical to optimize antibacterial management.

Table 3. Susceptibility of Key Pathogens in Acute Exacerbation of Chronic Bronchitis

Antibacterial Agent	*Streptococcus pneumoniae* (% resistant)†	*Haemophilus influenzae* (% resistant)†	*Moraxella catarrhalis* (MIC_{90})*
Penicillin	34	—	—
Ampicillin	—	27	8
Amoxicillin/clavulanate	7	—	—
Trimethoprim/sulfamethoxazole	32	22	25
Cefuroxime	25	1	2
Ceftriaxone	4	0	1.0
Azithromycin	28	1	0.03
Levofloxacin	1	1	0.06

*MIC_{90} indicates minimum inhibitory concentration for 90% of strains.
†Includes intermediate and resistant strains.
Adapted from reference 25.

MANAGEMENT

Numerous options are available for the management of AECB. Although not part of the acute man-

agement of AECB, none is more important on a long-term basis than a concerted effort to encourage the patient to stop smoking. In fact, the acute exacerbation might provide a "teachable moment" in which to reaffirm the smoking cessation message. In addition, pneumococcal vaccination and an annual influenza vaccination are essential for comprehensive care.

Goals

Successful management of AECB involves achievement of 3 goals:

- Quickly resolving the patient's symptoms
- Preventing relapse or lengthening the time between exacerbations
- Interrupting the vicious circle of recurrent infection and lung damage.

The methods used to achieve these goals depend on the severity of the exacerbation and the patient's risk factors.

Adjunctive Treatment

In addition to the use of antibiotics in appropriate patients, other treatments should be utilized:

- Removal of irritants
- Use of a bronchodilator
- Use of oxygen therapy.
- Hydration
- Use of a systemic corticosteroid
- Chest physical therapy.

Irritants should be removed or addressed if contributory. Because worsening airflow obstruction is characteristic in AECB, an increase in the bronchodilator dose or the temporary addition of a short-acting bronchodilator is critical to relax bronchial smooth muscle and reduce inflammation, thereby improving FEV_1. However, no incremental efficacy is achieved by adding an anticholinergic bronchodilator to a β-agonist bronchodilator (or vice versa) after achieving maximum bronchodilation. There is no difference in the efficacy of the β-agonists and the anticholinergic agent ipratropium in AECB. Methylxanthines, on the other hand, are less effective and are associated with more adverse effects than other bronchodilators. The few direct comparisons of metered-dose inhalers with nebulizers in AECB have generally shown similar efficacy.[16,19] Nonetheless, nebulizers may be preferred since drug deposition is unaffected during tachypnea.[19]

A systemic corticosteroid is beneficial in the case of significant pulmonary compromise, particularly if the patient requires hospitalization. The optimal dose and duration of therapy remain uncertain, although evolving data suggest that most of the improvement in lung function (as measured by FEV_1) occurs during the first 3 to 5 days of corticosteroid treatment.[19]

Another adjunctive therapy is oxygen. Although oxygen normally is administered by nasal prongs or face mask, administration by either mechanical ventilation or noninvasive positive pressure ventilation is appropriate if the patient is significantly hypoxemic or has a serum pH less than 7.3. Although *chronic* use of mucolytic drugs is of benefit in reducing the frequency of acute exacerbations and days of illness,[26] they are of no benefit in improving ventilatory function in AECB patients.[19]

While the few studies that have assessed the benefits of physical therapy during AECB have shown no significant benefit,[19] a recent study found that 2 approaches, oscillating positive expiratory pressure (using the FLUTTER device) and expiration with the glottis open in the lateral position (ELTGOL), were safe and effective in removing secretions without causing undesirable effects on oxygen saturation.[27]

Antibiotic Treatment

The role of an antibiotic in the management of AECB has been the subject of much research and discussion, but despite this, some uncertainty remains. This uncertainty may be due, at least in part, to the etiologic role of a virus in one third of AECB patients, a fact not taken into consideration in many (especially older) studies involving an antibacterial agent. Although few studies since 1980 have involved a placebo control, and the results have been conflicting,[13,28,29] the landmark study by Anthonisen et al demonstrated that certain patients treated with an antibacterial agent experienced faster resolution of symptoms and a higher success rate than patients treated with placebo.[13] Patients who experienced the most benefit were those with increased dyspnea, sputum volume, and sputum purulence; that is, sicker patients. Thus, patients in whom antibacterial therapy should be initiated are those with a documented history of chronic bronchitis who are thought to be experiencing an acute exacerbation and who have at least 2 of the following: increased dyspnea, increased sputum volume, and increased sputum purulence (Table 2).[9] The presence of at least 2 of these 3 symptoms presumably decreases the likelihood that a patient with a purely viral exacerbation would be treated with an antibacterial agent, because changes in sputum volume or purulence are less likely in a viral compared with a bacterial exacerbation, as discussed previously.

Several risk stratification strategies have been proposed, but none has been validated.[19] Nonetheless, evidence from many studies suggests that certain factors

Table 4. Factors Associated With an Increased Risk of Relapse

- Frequent purulent exacerbations
- Exacerbation within past 7 days
- Advanced age
- Poor functional status
- Comorbidities
- Chronic corticosteroid use
- Long duration of chronic obstructive lung disease
- Severe underlying lung function
- Malnutrition

increase the risk of relapse (**Table 4**).[9,19] Another important consideration in weighing the aggressiveness of therapy is the patient's ability to tolerate treatment failure given his or her respiratory status. Beyond patient morbidity and mortality, treatment failure has major economic consequences because the costs associated with hospitalization are the major determinant of the overall economic burden of AECB.[6]

For those patients with AECB in whom antibacterial therapy is appropriate, many agents are available. In selecting the agent to use, several factors can be considered.[30] Most agents used for AECB in the clinical setting are bactericidal and have a good safety profile. Therefore, spectrum of activity and resistance patterns, tracheobronchial penetration, and cost-effectiveness are the most important considerations. Penicillins and cephalosporins generally do not penetrate the tracheobronchial tree well.[30,31] As previously discussed, penicillins and first- and some second-generation cephalosporins (eg, cephalexin, cefaclor, cefuroxime) are beset by problems with resistance by the major pathogens. Fluoroquinolones and macrolides, on the other hand, do manifest good tracheobronchial penetration.[30] As a group, the respiratory-tract fluoroquinolones (eg, gatifloxacin, levofloxacin, moxifloxacin, sparfloxacin, trovafloxacin) are associated with a low level of resistance by *S pneumoniae*, *H influenzae*, and *M catarrhalis*, while more than 20% of *S pneumoniae* isolates are resistant to the macrolides (erythromycin, azithromycin, and clarithromycin).[32,33]

Numerous randomized, double-blind comparative clinical trials have been conducted over the past decade. Many within the past few years have involved a macrolide and/or a fluoroquinolone. For example, 11 of 13 studies compared ciprofloxacin or ofloxacin with agents such as amoxicillin, amoxicillin/clavulanate, clarithromycin, and cefuroxime axetil. The clinical success rate in the majority of these studies was at least 85% for both the fluoroquinolone and the comparator drug. Bacteriologic eradication also was at least 85% for the fluoroquinolone in the majority of studies. In fact, bacteriologic eradication was significantly greater with the fluoroquinolone than the comparator drug in 6 of 10 studies.[2]

More recently, moxifloxacin 400 mg orally once daily for 5 days was compared with azithromycin orally for 5 days (500 mg on the first day and 250 mg daily for 4 days).[34] The clinical resolution and bacteriologic eradication rates were equivalent. Azithromycin also has been compared with levofloxacin.[35] Patients received either azithromycin for 5 days (500 mg on the first day and 250 mg daily for 4 days) or levofloxacin 500 mg orally daily for 7 days. Again, the clinical resolution and bacteriologic eradication rates were equivalent. A 5-day course of levofloxacin also has been shown to yield clinical success and bacteriologic eradication rates equivalent to those of a 7-day course of levofloxacin.[36] Other presumed benefits include reduced cost and improved medication adherence.

Side effects of most of the agents are well established; the most common, which are relatively minor, primarily involve the gastrointestinal tract.[19,30,31] There are some notable exceptions, however. The incidence of diarrhea associated with amoxicillin/clavulanate is 9% with standard doses, but is increased to about 15% with the high doses needed for sicker patients, as discussed below.[37] Temafloxacin is no longer available, and the use of sparfloxacin is limited. Similarly, the use of trovafloxacin is severely restricted because of rare but severe liver toxicity, and grepafloxacin is no longer available in the United States due to the rare occurrence of torsades de pointes.[31] Other fluoroquinolones that are associated with QTc prolongation include sparfloxacin, gatifloxacin, and moxifloxacin.[2,31] Their use in patients with severe underlying heart disease, severe bradycardia, or uncorrected hypokalemia, and in those receiving class IA or class III antiarrhythmic agents should be avoided. The macrolide clarithromycin also is associated with QTc prolongation when given in combination with pimozide or terfenadine.[38]

Some fluoroquinolones have been associated with alterations in serum insulin and glucose levels. Clinical investigation in healthy adults has shown that multiple-dose gatifloxacin causes a transient increase in serum insulin 1 hour after administration with no alteration of glucose tolerance, pancreatic β-cell function, or predose fasting serum glucose level.[39] Recently, several

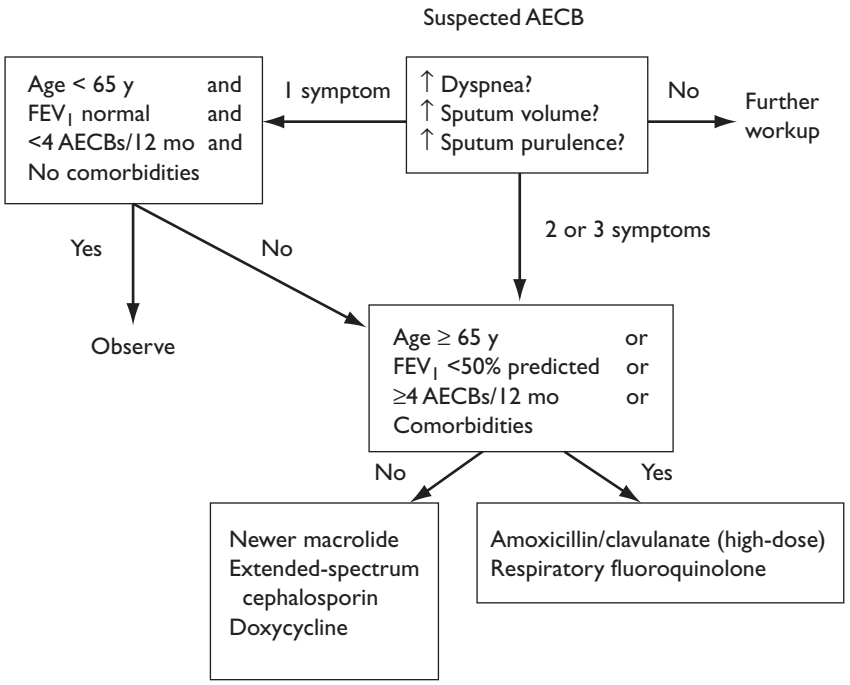
case reports of gatifloxacin-induced hypoglycemia in patients with type 2 diabetes mellitus treated with various hypoglycemic agents have been published. Patient symptoms ranged from asymptomatic hypoglycemia to severe symptomatic hypoglycemia with a seizure.[40-42]

The clinical and economic implications of antibacterial selection have been assessed in 60 outpatients who had a total of 224 episodes of AECB.[43] Patients were divided into 3 groups based on the antibacterial agent they received. Group 1 patients received amoxicillin, trimethoprim/sulfamethoxazole, tetracycline, or erythromycin; group 2 received cephradine, cefuroxime, cefaclor, or cefprozil; and group 3 received amoxicillin/clavulanate, azithromycin, or ciprofloxacin. Group 1 patients failed to respond to therapy significantly more frequently than those in group 3 (18.0% vs 5.3%). The time between subsequent AECB episodes that required treatment was significantly longer for group 3 compared with groups 1 and 2. Although the drug cost was lowest for group 1 and highest for group 3, the mean total cost of AECB treatment was lowest for patients in group 3 compared with groups 1 and 2. A significant factor in the lower total cost was a significant reduction in the need for hospitalization in group 3.

Finally, a simplified risk stratification and antibacterial management algorithm is suggested based on the prevailing data (**Figure 4**). All patients should receive adjunctive care, which might include a bronchodilator, oxygen, a corticosteroid, hydration, and chest physical therapy, as well as therapies for comorbidities as appropriate. A patient who presents with either increased dyspnea, increased sputum volume, or increased sputum purulence and who has none of the 4 risk factors (age > 65 years, FEV_1 < 50% of the predicted value, ≥4 AECBs in 12 months, 1 or more comorbidities) can be observed and managed with adjunctive treatment. A patient who has 1 of the 3 primary symptoms and who has at least 1 of the 4 risk factors is considered at some risk and requires antibacterial treatment. Of these patients, those who have some impairment of lung function but have an FEV_1 that is ≥50% of the predicted value (without being normal) are considered at some risk and should be treated with a newer macrolide, extended-spectrum cephalosporin, or doxycycline. The extended-spectrum cephalosporins may have advantages of improved efficacy and safety compared with the first-generation cephalosporins cephalexin and cefaclor. Doxycycline is a good choice if *M catarrhalis* alone is suspected based on the patient's history, or as an alternative to those allergic to the newer macrolides and cephalosporins.[31] For a patient thought to be infected with an atypical bacterium (again based on the patient's history), a macrolide or a respiratory fluoroquinolone should be used.

The majority of patients, however, will be sicker and will have 1 or more of the 4 risk factors. As they are considered at high risk, they should receive high-dose amoxicillin/clavulanate or a respiratory fluoroquinolone to cover the most common typical and atypical pathogens. It is important to note that the dose of amoxicillin/clavulanate is 875 mg twice daily or 500 mg three times daily. The choice of an antibacterial agent should, of course, be altered based on local susceptibil-

Figure 4. Risk Stratification and Antibacterial Management Algorithm*



AECB indicates acute exacerbation of chronic bronchitis; FEV_1, forced expiratory volume in 1 second.
*Note that all patients with 1 or more symptoms also require supportive care (eg, bronchodilator, steroid, oxygen).
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ity patterns. Additionally, patients who do not respond within 24 to 36 hours should be reevaluated. Antibiotic treatment should be continued until the patient's condition has returned to baseline, generally 5 to 10 days depending on the antibiotic used.

SUMMARY

Chronic bronchitis is characterized by 1 or more key symptoms: increased sputum production, increased sputum purulence, and/or worsening dyspnea. Eighty percent of the cases of AECB are due to infection, with half due to aerobic bacteria. There has been increasing antibacterial resistance by the 3 most prevalent pathogens (*S pneumoniae*, *H influenzae*, and *M catarrhalis*). Treatment of AECB is based on the number of key symptoms and risk factors that are present. Only patients with at least 1 key symptom and 1 risk factor should be treated with an antibacterial. A newer macrolide, extended-spectrum cephalosporin, or doxycycline is appropriate for an exacerbation of moderate severity, while high-dose amoxicillin/clavulanate or a respiratory fluoroquinolone should be used for a severe exacerbation. All patients should receive supportive care.

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