

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

**MODERN PRACTICE
OF INTERNAL MEDICINE
WITH EMERGENCY CONDITIONS**

**Management of the patient
with chronic cough**

Guidelines for students and interns

**Сучасна практика
внутрішньої медицини
з невідкладними станами**

Ведення хворих з хронічним кашлем

*Методичні вказівки
для студентів та лікарів-інтернів*

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Management of the patient with chronic cough

General Outcome

The students should be able to describe main links of pathogenesis, clinical features, diagnostic and treatment of chronic cough.

The aim of this topic is to provide the student with an opportunity to:

- Provide a basic overview of the pathophysiology, diagnosis, and classification of chronic cough
- Evaluate guideline-based management strategies for the treatment of chronic cough.
- Develop an individualized pharmacotherapy and monitoring plan for the management of chronic cough, when given specific patient information.

Specific Learning Outcomes:

Upon successful completion of this unit, the students should be able to:

1. Describe chronic cough classifications.
2. Describe the main mechanism of chronic cough.
3. Describe the main clinical features of chronic cough.
4. List and describe the group of drugs that are used in the treatment of chronic cough and give specific examples of each.
5. Make a treatment plan of patient with chronic cough.

Specification of the theoretical question for training of "Management of the patients with chronic cough"

Student must know:

1. What is the definition of chronic cough?
2. What are the main causes of chronic cough?
3. What are the main pathogenetic links of chronic cough?
4. What are the main types of chronic cough?
5. What laboratory tests are used in patients with chronic cough?
6. What imaging studies are used in patients with chronic cough?
7. What treatment methods are used in patients with chronic cough?

DEFINITION

Cough is the most common symptom of respiratory disease. It is caused by stimulation of sensory nerves in the mucosa of the pharynx, larynx, trachea and bronchi. Acute sensitisation of the normal cough reflex occurs in a number of conditions, and it is typically induced by changes in air temperature or exposure to irritants such as cigarette smoke or perfumes.

Results of epidemiologic surveys suggest that patients who seek medical care for a cough account for only a small part of the population with this symptom. Chronic cough can be associated with significant distress and impairment in quality of life.

CLASSIFICATION

Cough can be divided into three categories: acute (i.e., lasting less than three weeks), subacute (i.e., lasting three to eight weeks), and chronic (i.e., lasting longer than eight weeks).

ETIOPATHOGENESIS

The cough reflex is triggered by mechanical or inflammatory changes or irritants in the airways. The afferent pathway is via the vagus nerve to respiratory neurones termed the "cough centre" in the brainstem. Higher cortical centres also control the cough. Chronic cough tends to be inhibited during sleep.

Chronic cough is often associated with bronchial hyperreactivity (bronchial hyper-responsiveness), which can persist in the absence of the initiating cough event. Bronchial hyper-responsiveness is defined as a state of increased sensitivity to a wide variety of airway narrowing stimuli, eg exercise, dry or cold air, hypertonic or hypotonic aerosols. It occurs in asthma and chronic obstructive pulmonary disease (COPD), but also can occur in the absence of lung disease.

Most cases of troublesome cough reflect the presence of an aggravant (asthma, drugs, environmental, gastro-esophageal reflux, upper airway pathology) in a susceptible individual. The most common causes of chronic cough, other than smoking in adults, are postnasal drip, asthma, and gastro-esophageal reflux disease (GERD).

Common causes

Smoking (active or passive).

Asthma (and its variants, ie cough variant asthma, eosinophilic bronchitis) – all of which are steroid-responsive.

COPD.

GERD.

Postnasal drip.

Environmental pollution.

Less common causes

Cardiovascular – left ventricular failure, pulmonary emboli, aortic aneurysm.

Chronic infections – bronchiectasis, tuberculosis, cystic fibrosis, lung abscess.

Postinfectious cough – may be more likely following infection with Mycoplasma pneumoniae, chlamydial pneumonia and whooping cough.

Parenchymal lung diseases – interstitial lung fibrosis, emphysema, sarcoidosis.

Tumours – lung cancer, metastatic carcinoma, lymphoma, mediastinal tumours, benign tumours.

Upper airway conditions (other than chronic rhinitis, above) – chronic tonsil enlargement, obstructive sleep apnoea, chronic snoring, irritation of external auditory meatus. Laryngeal problems are increasingly recognised as being part of chronic cough.

Foreign body in large airways – recurrent aspiration, inhaled foreign body, endobronchial sutures.

Rarely, cough may be due to cardiac arrhythmias.

Cough only when supine – may be due to collapse of large airways.

Diffuse panbronchiolitis – a recognised cause in Japan, responds to low-dose macrolide antibiotics (but resistant to steroids).

Chronic cough may be a presentation of a complex involuntary tic (eg as sometimes seen in Tourettes Syndrome).

One small study suggested that vitamin B12 deficiency contributes to chronic cough (perhaps due to sensory neuropathy).

Idiopathic or psychogenic – a diagnosis of exclusion.

Angiotensin-converting enzyme (ACE) inhibitors.

Occupational exposure to irritants (including farm workers, workers exposed to hot acidic conditions in a bottle factory, and workers exposed to hot chilli peppers).

Whooping cough – in young adults and may be more common than previously supposed.

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APPROACH TO THE PATIENT

A detailed *history* frequently provides the most valuable clues for the etiology of the cough. Particularly important questions include:

1. Is the cough acute, subacute, or chronic?
2. At its onset, were there associated symptoms suggestive of a respiratory infection?
3. Is it seasonal or associated with wheezing?
4. Is it associated with symptoms suggestive of postnasal drip (nasal discharge, frequent throat clearing, a "tickle in the throat") or gastroesophageal reflux (heartburn or sensation of regurgitation)? However, the absence of such suggestive symptoms does not exclude either of these diagnoses.
5. Is it associated with fever or sputum? If sputum is present, what is its character?

6. Does the patient have any associated diseases or risk factors for disease (e.g., cigarette smoking, risk factors for infection with HIV, environmental exposures)?

7. Is the patient taking an ACE inhibitor?

Threatning symptoms:

- Cough with increasing intensity that lasting above week.
- Cough accompanied by long steading (weeks) hyperthermia 38 °C.
- Cough accompanied by hyperthermia above 38 °C during 3 days or more.
- Cough accompanied by dyspnea and thorasic pain on breathing.
- Cough of pus.
- Blood spitting.
- Cough with pronounced dyspnea.
- Cough and weakness and weight loss.
- Excessive sweating, shivering.
- Sudden attack of severe cough.
- Severe cough during an hour without any interval.
- Abundant expectoration of sputum.

**Potentially serious causes of a chronic cough
(duration of 8 weeks or more) in an adult and suggestive features
on history and physical examination**

Condition	Suggestive Clinical Features
Asthma	Wheezing, triggers such as exercise, cold air
Tuberculosis	Fever, weight loss, night sweats, hemoptysis, from an endemic area
Primary lung cancer	Weight loss, hemoptysis, smoking history, older age
Metastases to lungs	History of cancer
Heart failure	History of cardiac disease, dyspnea, orthopnea, dependent edema
Chronic obstructive pulmonary disease	Smoking history, chronic sputum production
Interstitial lung disease	Dyspnea, possible environmental exposure, inspiratory crackles present on lung examination

The general *physical examination* may point to a systemic or non-pulmonary cause of cough, such as heart failure or primary nonpulmonary neoplasm. Examination of the oropharynx may provide suggestive evidence for postnasal drip, including oropharyngeal mucus or erythema, or a "cobblestone" appearance to the mucosa. Auscultation of the chest may demonstrate inspiratory stridor (indicative of upper airway disease), rhonchi or expiratory wheezing (indicative of lower airway disease), or inspiratory crackles

(suggestive of a process involving the pulmonary parenchyma, such as interstitial lung disease, pneumonia, or pulmonary edema).

Chest radiography may be particularly helpful in suggesting or confirming the cause of the cough. Important potential findings include the presence of an intrathoracic mass lesion, localized pulmonary parenchymal opacification, or diffuse interstitial or alveolar disease. An area of honey-combing or cyst formation may suggest bronchiectasis, while symmetric bilateral hilar adenopathy may suggest sarcoidosis.

Pulmonary function testing is useful for assessing the functional abnormalities that accompany certain disorders producing cough. Measurement of forced expiratory flow rates can demonstrate reversible airflow obstruction characteristic of asthma. When asthma is considered but flow rates are normal, bronchoprovocation testing with methacholine or cold-air inhalation can demonstrate hyperreactivity of the airways to a bronchoconstrictive stimulus. Measurement of lung volumes and diffusing capacity is useful primarily for demonstration of a restrictive pattern, often seen with any of the diffuse interstitial lung diseases.

If *sputum* is produced, gross and microscopic examination may provide useful information. Purulent sputum suggests chronic bronchitis, bronchiectasis, pneumonia, or lung abscess. Blood in the sputum may be seen in the same disorders, but its presence also raises the question of an endobronchial tumor. Greater than 3 % eosinophils seen on staining of induced sputum in a patient without asthma suggests the possibility of eosinophilic bronchitis. Gram and acid-fast stains and cultures may demonstrate a particular infectious pathogen, while sputum cytology may provide a diagnosis of a pulmonary malignancy.

More specialized studies are helpful in specific circumstances.

Fiberoptic bronchoscopy is the procedure of choice for visualizing an endobronchial tumor and collecting cytologic and histologic specimens. Inspection of the tracheobronchial mucosa can demonstrate endobronchial granulomas often seen in sarcoidosis, and endobronchial biopsy of such lesions or transbronchial biopsy of the lung interstitium can confirm the diagnosis. Inspection of the airway mucosa by bronchoscopy may also demonstrate the characteristic appearance of endobronchial Kaposi's sarcoma in patients with AIDS.

High-resolution computed tomography (HRCT) can confirm the presence of interstitial lung disease and frequently suggests a diagnosis based on the specific abnormal pattern. It is the procedure of choice for demonstrating dilated airways and confirming the diagnosis of bronchiectasis.

TREATMENT OF COUGH

Anti-Tussive Drugs

• Central Antitussives

Mainly suppress cough centre in medulla (both central & peripheral effects) E.g., Opioid drugs (codeine, pholcodeine, noscapine, dextromethorphan). Opioids are believed to inhibit cough primarily by their effect on the cough center; opiate anti-tussives have a greater adverse side effect profile. Because of the potential for abuse and addiction with opioids, nonopioid antitussives (e.g., dextromethorphan) are preferred in the treatment of acute cough. They are widely available without prescription and thus classified as over-the-counter (OTC) drugs.

Codeine

- An opium alkaloid.
- It is more selective for cough centre.
- Centrally acting anti tussives increase threshold for cough.
- Suppresses cough for about 6 hours.
- The antitussive action is blocked by naloxone indicating that its action may be exerted through Opioid receptors in the brain.
- Cough suppression occur with low doses of opioids than those needed for analgesia.(sub-analgesic dose 15 mg).
- Abuse liability is low, but present.
- Adverse Effects: constipation; respiratory depression & drowsiness; driving may be impaired.
- Contraindicated in asthmatics & in patients with diminished respiratory reserve.

• Peripheral Antitussives

Regarding peripherally acting anti-tussives, levodropropizine, which is an orally-administered non-opiod agent whose peripheral anti-tussive action may result from its modulation of sensory neuropeptide levels within the respiratory tract.

• Expectorants (Mucokinetics)

Expectorants are defined as medications that improve the ability to expectorate purulent secretions. This term is now taken to mean medications that increase airway water or the volume of airway secretions, including secretagogues that are meant to increase the hydration of luminal secretions (eg, hypertonic saline or mannitol) and abhesives that decrease the adhesivity of secretions and thus unstick them from the airway (eg, surfactants). Expectorants do not alter ciliary beat frequency or mucociliary clearance. Oral expectorants were once thought to increase airway mucus secretion by acting on the gastric mucosa to stimulate the vagus nerve, but that is probably inaccurate. The most commonly used expectorants are simple

hydration, including bland aerosol, oral hydration, iodide-containing compounds such as super-saturated potassium iodide or iodinated glycerol, glyceral guaiacolate (guaifenesin), and the more recently developed ion-channel modifiers such as the P2Y2 purinergic agonists.

Guaifenesin is an expectorant, the action of which promotes or facilitates the removal of secretions from the respiratory tract. By increasing sputum volume and making sputum less viscous, guaifenesin facilitates expectoration of retained secretions. Helps loosen phlegm (mucus) and thin bronchial secretions to rid the bronchial passageways of bothersome mucus, drain bronchial tubes, and make coughs more productive. Helps loosen phlegm and thin bronchial secretions in patients with stable chronic bronchitis. Adults and children 12 years of age and older: Two to four teaspoonfuls (200 to 400 mg) every four hours, not to exceed 2 400 mg (24 teaspoonfuls) in 24 hours.

• Mucolytics

Help in expectoration by liquefy the viscous tracheobronchial secretions.
E.g., *Bromhexine, Acetylcysteine*.

Bromhexine

➤ Synthetic derivative of vasicine (alkaloid= *Adhatoda vasica*). It is secretolytic, increasing the production of serous mucus in the respiratory tract and makes the phlegm thinner and less viscous. This contributes to a secretomotoric effect by helping the cilia transport the phlegm out of the lungs. It has been shown to increase the proportion of serous bronchial secretion, making it more easily expectorated. It is indicated as "secretolytic therapy in bronchopulmonary diseases associated with abnormal mucus secretion and impaired mucus transport".

➤ Bromhexine is contained in various formulations, high and low strength syrups 8 mg/5 ml, 4 mg/5 ml, tablets and soluble tablets (both with 8 mg bromhexine) and solution for oral use 10 mg/5 ml, adapted to the need of the patients.

➤ Adverse effects: nausea, vomiting, bronchospasm in bronchial asthma.

Ambroxol

➤ Ambroxol is a clinically proven systemically active mucolytic agent. When administered orally onset of action occurs after about 30 minutes. The breakdown of acid mucopolysaccharide fibers makes the sputum thinner and less viscous and therefore more easily removed by coughing. Although sputum volume eventually decreases, its viscosity remains low for as long as treatment is maintained.

Indications.

- All forms of tracheobronchitis, emphysema with bronchitis pneumoconiosis, chronic inflammatory pulmonary conditions, bronchiectasis, bronchitis with bronchospasm asthma. During acute exacerbations of bronchitis it should be given with the appropriate antibiotic.

Contraindications

- There are no absolute contraindications but in patients with gastric ulceration relative caution should be observed.

- Adults: daily dose of 30 to 120 mg taken in 2 to 3 divided doses.

Acetylcysteine (N-acetylcysteine or N-acetyl-L-cysteine (NAC)

Acetylcysteine acts to reduce mucus viscosity by splitting disulfide bonds linking proteins present in the mucus (mucoproteins). Furthermore, with respect to its use as a mucolytic agent in patients with COPD, it is hypothesized that acetylcysteine may exert additional beneficial effects through its anti-inflammatory and antioxidant properties.

Inhaled acetylcysteine is indicated for mucolytic ("mucus-dissolving") therapy as an adjuvant in respiratory conditions with excessive and/or thick mucus production. Such conditions include emphysema, bronchitis, tuberculosis, bronchiectasis, amyloidosis, pneumonia, cystic fibrosis, chronic obstructive pulmonary disease, pulmonary fibrosis, and inhalation injury in children who have burns. It is also used post-operatively, as a diagnostic aid, and in tracheotomy care. It may be considered ineffective in cystic fibrosis.

Acetylcysteine is available in different dosage forms for different indications:

Solution for inhalation (Assist, Mucomyst, Mucosil) – inhaled for mucolytic therapy or ingested for nephroprotective effect (kidney protection).

Intravenous injection (Assist, Parvolex, Acetadote) – treatment of paracetamol/acetaminophen overdose.

Oral solution – various indications.

Effervescent tablets.

Ocular solution – for mucolytic therapy.

Tablets – sometimes in a sustained release formula sold as a nutritional supplement.

- **Antihistamines.** *Chlorpheniramine, diphenhydramine, promethazine*

- Added to antitussives/expectorant formulation.
- Due to sedative & anticholinergic actions produce relief in cough but lack selectivity for cough centre.
- No expectorant action = decrease secretions (anticholinergic effect).
- Suitable for allergic cough (not for asthma).

- Bronchodilators

- Bronchospasm or stimulation of pulmonary receptors = induce or aggravate cough + bronchoconstriction e.g. β_2 -agonist (*salbutamol, terbutaline*).
 - MOA of bronchodilators in cough.
 - Increase surface velocity of air flow during cough → Clear secretions of airway.
 - Not used routinely for every type of cough but only when bronchoconstriction is present.

- Pharyngeal Demulcents

- Soothe the throat (directly & also by promoting salivation).
- Decrease afferent impulses from inflamed/irritated pharyngeal mucosa.
- Provide symptomatic relief in dry cough arising from throat.

E.g. *lozenges, cough drops, glycerine, liquorice, honey*.

WORKUP AND MANAGEMENT

Antitussives, such as codeine and dextromethorphan, have been shown to have limited or no efficacy in the treatment of chronic cough and any beneficial effect is largely due to placebo effect. As such, the clinician should try to elucidate and identify the underlying cause of the cough to effectively manage it.

Every patient with chronic cough needs a thorough history taken and physical examination performed as part of their evaluation. Each patient should also have a chest radiograph taken.

Surprisingly, the medical history (in terms of the patient's description of the character, timing, and presence or absence of sputum production) has been shown to have little or no diagnostic value. What is of value from the medical history is whether or not the patient is or has been a smoker; is taking an ACE inhibitor; is living in a geographic area where tuberculosis or certain fungal diseases are endemic; has any systemic symptoms, a history of cancer, tuberculosis, or AIDS; or has a large pulmonary mass visible on chest radiograph.

Management should begin with cessation of smoking or ACE inhibitor use in those patients whose history indicates such action. Most patients have a resolution of their cough within 4 weeks of smoking cessation. Cough related to ACE inhibitor use usually subsides within 2 weeks.

If the chest radiograph findings are abnormal, further workup depends on the specific finding. Chest CT scan, bronchoscopy, needle biopsy, and sputum studies are all potentially warranted studies if a pulmonary lesion is found.

For the immunocompetent nonsmoker who does not use ACE inhibitors and has normal chest radiograph findings, a systematic approach to the most common causes of chronic cough is warranted, keeping in mind that more than one cause may be present. The body of literature regarding

specific treatments and the expected time frame of response is extensive, and the accuracy of the diagnosis is confirmed by the patient's response to these treatments. From both theoretical and cost effectiveness standpoints, empiric treatment of the 3 most common causes of cough is favored over extensive testing at the outset. Further, sequential and additive therapy may be needed because more than one cause of cough is often present.

Upper airway cough syndrome

Because upper airway cough syndrome (UACS) is the most common cause of chronic cough, it should be treated first. In patients in whom the cause of the UACS-induced cough is apparent, specific therapy directed at this condition should be instituted. This includes avoiding environmental irritants and offending antigens, treating sinusitis with antibiotics, and weaning patients off nasal decongestants for rhinitis medicamentosa. Further workup may include allergy testing for allergic rhinitis or sinus CT scan for sinusitis, as indicated.

For patients in whom the cause is not apparent, empiric therapy should be instituted with a combination of an antihistamine and decongestant. First-generation antihistamines such as azatadine and dexbrompheniramine plus pseudoephedrine have shown more effectiveness than newer, less-sedating antihistamines. Patients typically respond within 2 weeks of initiating therapy but may sometimes take several months.

Asthma

Asthma should be considered only after the UACS evaluation and empirical treatment trial are complete. Ideally, patients should undergo spirometry and bronchoprovocation challenge with methacholine, which reveals reversible airflow obstruction. The negative predictive value for a negative challenge approaches 100 %.

The initial treatment of asthma consists of beta₂-agonists and inhaled corticosteroids (ICS) and response is usually seen within 1 week, with complete resolution taking up to 8 weeks. Some patients may require a trial of oral corticosteroids before a response is seen. However, because leukotriene inhibitors have been shown to be effective in patients with asthma-induced cough, they should be tried prior to oral corticosteroid therapy.

Nonallergic eosinophilic bronchitis

Because its diagnosis is made easily, nonallergic eosinophilic bronchitis (NAEB) is the next etiology to consider, even though GERD is more common. An induced sputum test that reveals increased eosinophils is the diagnostic procedure of choice. Treatment includes ICS, with oral corticosteroids reserved for refractory cases. Response is usually seen within 4 weeks.

Gastroesophageal reflux disease

Cough may be related to distal reflux at the lower esophageal sphincter, or due to micro-aspiration of acid into the trachea. There may be associated

esophageal dysmotility. lower esophageal sphincter reflux is often long-standing and is associated with a productive or non-productive daytime cough and minimal nocturnal symptoms. It is worse after meals and when sitting down, due to increased intraabdominal pressure being transmitted to the lower esophageal sphincter. Micro-aspiration is associated with more prominent symptoms of reflux or dyspepsia, although these are not always present. Patients may have an intermittent hoarse voice, dysphonia, and sore throat.

Laryngoscopy may reveal posterior vocal cord inflammation, but this is not a reliable sign.

A trial of treatment for both is recommended. This is with a high-dose proton pump inhibitor for at least 3 months, although longer treatment may be required to control cough. H2 receptor blockers are also effective and pro-kinetics like metoclopramide may help. Other reflux avoidance measures should be carried out: avoiding caffeine, wearing loose fitting clothes, sleeping with an empty stomach (avoid eating <4 hours before bed), sleeping propped up.

Investigation, if required, due to treatment failure or diagnostic uncertainty, is with 24-hour ambulatory pH monitoring, which determines the presence of reflux events. These may not necessarily be responsible for the cough, so it is not a very specific or sensitive test. Esophageal manometry can be used to measure the lower esophageal sphincter pressure and esophageal contractions after swallowing to determine the presence of esophageal dysmotility.

Cough and Lung Cancer

Lung cancer is the etiology in less than 2 percent of the cases of chronic cough but is deadly to the patient and to the doctor if undetected – CT scan, when? Most cases of lung cancer that manifest with cough are due to neoplasms originating in the large central airways.

Bronchogenic cancer should be considered as a possible etiology of cough in any current or former smoker, and should be particularly suspected in those with:

- A new cough or a recent change in chronic "smoker's cough".
- A cough that persists more than one month following smoking cessation.
- Hemoptysis that does not occur in the setting of an airway infection.

Further workup

Only when management of the most common causes has failed to yield a resolution of cough should a more extensive workup begin. This can include induced sputum testing for acid fast bacillus, high resolution CT scan of the chest, and bronchoscopy. Oftentimes, these tests should be performed by a cough specialist. If further testing does not reveal the cause, then the patient most likely has chronic cough hypersensitivity syndrome. Owing to

inflammation and hyperresponsiveness of the airway from some inciting cause, tissue remodeling has occurred, leading to an enhanced cough reflex that maintains the cough even though the inciting cause has resolved. In these truly idiopathic cases, therapeutics are limited, but ongoing research is focusing on medicines that either directly or indirectly affect the cough reflex.

UPPER AIRWAY COUGH SYNDROME – INDUCED CHRONIC COUGH

Patients with chronic cough should first be treated with a first-generation antihistamine/decongestant. If the patient has complete or partial resolution of cough after one to two weeks of antihistamine/decongestant therapy, then it is assumed that upper airway cough syndrome was the cause and therapy should be continued. If the patient has persistent nasal symptoms, it is appropriate to begin a topical nasal steroid. If symptoms still persist, it is an indication for sinus imaging.

Patients with mucosal thickening should be treated for sinusitis. If the patient does not respond to sinusitis therapy, she or he should be referred to an ear, nose, and throat specialist.

If the patient has only partial resolution of cough but no signs of upper airway cough syndrome, then an evaluation for asthma should be done.

ASTHMA-INDUCED CHRONIC COUGH

It has been shown that bronchoprovocation is useful in the evaluation for cough caused by asthma. A positive challenge usually warrants trial treatment for asthma and clinical monitoring. The majority of patients will respond to treatment with inhaled corticosteroids and beta agonists after one week of therapy; it may take up to eight weeks for complete cough resolution.

In patients who do not respond or cannot take inhaled medication, treatment with oral corticosteroids for five to 10 days is an option. Because oral leukotriene inhibitors may be effective, consideration should be given to adding a leukotriene inhibitor before an oral corticosteroid.

If treatment for upper airway cough syndrome and asthma have both failed, nonasthmatic eosinophilic bronchitis should be considered next.

NONASTHMATIC EOSINOPHILIC BRONCHITIS – INDUCED CHRONIC COUGH

To diagnose nonasthmatic eosinophilic bronchitis, an induced-sputum test should be performed to determine if the patient has an increased number of eosinophils. If the patient appears to have nonasthmatic eosinophilic bronchitis, treatment with inhaled corticosteroids is recommended. Cough should resolve within four weeks of therapy.

Short-acting inhaled beta2-agonist (chronic stable disease or acute exacerbation).

Ipratropium (chronic stable disease or acute exacerbation).
Long-acting beta₂ agonist (e.g., salmeterol) plus inhaled cortico-steroid (chronic stable disease).
Consider theophylline (chronic stable disease only).
Codeine or dextromethorphan (short term for bothersome cough).

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