

Asthma in the Elderly



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KEYWORDS

• Aging • Asthma • Lung function • Overlap syndrome • Phenotypes

KEY POINTS

- New-onset asthma may occur at any age.
- Elderly patients with long-standing asthma usually have a history of atopy; IgE levels are elevated and often there is a history of allergic rhinitis.
- Asthma that begins at an advanced age is usually nonatopic and has low rates of remission.
- There are no specific protocols for the treatment of asthma in the elderly; treatment entails objective monitoring, avoidance of asthma triggers, pharmacotherapy, and patient education.
- Most elderly asthmatics tolerate asthma medications well with minimal adverse drug reactions.

INTRODUCTION

Asthma was long considered a childhood disease. There was little focus on asthma in elderly subjects until the Tucson epidemiologic study of obstructive lung disease brought attention to asthmatics in this age group.¹ This longitudinal study began in 1971 and results of the eighth survey in 1984 showed that active asthma is relatively common in people over age 65 years of age. Many subjects had severe disease with marked ventilatory impairment and suffered with this disabling disorder for years. Fewer than 1 of 5 in the study group went into complete remission and the death rates in the asthmatics tended to be higher than in nonasthmatics.² A further report of patients who developed asthma after the age of 60 showed that the serum IgE level was closely related to the likelihood of a subsequent asthma diagnosis and that a rapid decrease in lung function often occurred around the time of the initial diagnosis.³

The Tucson epidemiologic study offered the first characterization of asthma in the elderly (AIE) and highlighted the need for further investigation in the group. In the decades that followed, it became apparent that AIE is underdiagnosed and undertreated and that there is a paucity of knowledge regarding many aspects of the disease in this

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age group.^{4–11} The need for an assessment of current knowledge became apparent. This resulted in a workshop convened by the National Institute on Aging in September 2008, to improve the understanding and care of AIE, defined as asthma in those over 65 years of age.¹² At that time, it was acknowledged that at least 2 phenotypes exist among elderly asthmatics, namely, those with long-standing asthma have more severe airflow limitation and less complete reversibility than those with late-onset asthma that often, as was seen in the Tucson study, can occur for the first time at an advanced age.^{1,13} This raised questions regarding the pathophysiological mechanisms of AIE. Are they different from those seen in young asthmatics? And are aging-related changes in respiratory and immune physiology affecting the manifestations of this disease in the elderly?

It was acknowledged at the National Institute on Aging workshop that asthma presenting at an advanced age often has similar clinical and physiologic consequences as seen with younger individuals, but there are a number of confounding influences that complicate asthma in this age group, including the presence of comorbid illnesses. In addition, the clinical presentation may be highly variable as a result of the heterogeneity among older people that ranges from very “fit” to very “frail.” In May 2015, an American Thoracic Society Workshop on Asthma in the Elderly was convened to review the current state-of-the-art knowledge and provide future directions for research.¹⁴

EPIDEMIOLOGY

The most recent revised global estimate of asthma from the World Health Organization suggests that as many as 334 million people have asthma, and that the burden of disability is high.¹⁵ Asthma is especially seen with increased urbanization and with aging populations. A cohort of asthmatics in Norway followed for 11 years showed a higher risk for older patients. The incidence of asthma per 1000 person years was 2.4, 3.1, and 5.4 for age groups 15 to 29, 30 to 45, and 50 to 70 years respectively.¹⁶ Similar estimates from the United States, Poland, and Sweden have been reported.¹⁶ The incidence of this disease has increased substantially in recent decades.^{16,17} About 250,000 asthmatics die from this disease each year, often as a result of poor access to care, poor environmental control, and a lack of proper medications.¹⁸

A comprehensive epidemiologic study from the United States National Surveillance of Asthma was conducted for several decades and has been reviewed in the American Thoracic Society workshop document.¹⁷ The data on Americans across the lifespan, including those age 65 years of age and older, showed that older Americans had (1) the greatest increase in the prevalence of current asthma, from 6.0% in 2001 to 8.1% in 2010, (2) the highest rate of asthma-related deaths, and (3) the second highest rate of asthma-based physician office visits and hospitalizations, but (4) the lowest rate of having an asthma attack and an asthma-based emergency department visit, respectively. The most vulnerable of the older Americans were females, African Americans, Hispanics, and low-income groups.

In an analysis of a large US database of emergency department visits and hospitalizations for asthma between 2006 and 2008, Tsai and colleagues¹⁹ found that patients over age 65 with asthma had higher rates and greater durations of hospitalization, greater mortality, and more near-fatal attacks.

The presence of age-associated comorbidities of AIE such as heart disease, chronic obstructive pulmonary disease (COPD), kyphosis, gastroesophageal reflux, and pulmonary vascular disease makes determining asthma-related health care use difficult. In addition, many common geriatric conditions such as cognitive impairment, falls, low

body mass index, dizziness, and vision impairment can have a significant effect on activities of daily living and these conditions can be as prevalent as common chronic diseases in the elderly.²⁰

PATHOPHYSIOLOGY

Asthma is caused by a complex interaction of cells, mediators, and cytokines that result in airway inflammation.^{21,22} Inflammatory cells such as mast cells, eosinophils, activated T lymphocytes, and neutrophils can be seen on bronchial biopsies of asthmatic patients. Their release of specific cytokines seems to direct the movement of cells to the site of airway inflammation. Inflammatory cytokines also activate cells, causing them to further release their mediators. Mast cells, usually as a result of IgE-mediated stimulation, release preformed mediators, such as histamine, leukotrienes, prostaglandins, and proteases that promote bronchoconstriction, airway edema, mucus hypersecretion, and vascular congestion in the airways. Eosinophil infiltration is prominent in most asthmatics. Denudation of the airway epithelium can occur in severe asthma. This process can lead to airway edema and loss of substances in the mucosa that protect the airway. Epithelial damage promotes bronchial hyperresponsiveness (bronchoconstrictive responses). Increased access to sensory nerve endings may contribute to this phenomenon. Another characteristic finding in severe asthma is the presence of tenacious mucus plugs in the airways. Death from asthma usually occurs from blockage of the airways by diffuse mucus plugging. Thickening of the reticular basement membrane, the laminar reticularis, is observed on light microscopy and is a constant feature of asthma. There is also evidence of increased bronchial smooth muscle mass (hypertrophy and hyperplasia) that contributes considerably to the thickness of the airway wall. The airway architecture is also changed by the deposition of types I, III, and V collagen and fibronectin beneath the basement membrane. This has been referred to as “subepithelial fibrosis.” These architectural changes collectively seen in the airways of many elderly (and some younger) asthmatics are referred to as airway remodeling and are thought to cause permanent changes that result in fixed airflow obstruction. The degree of remodeling seems to worsen with more severe degrees of asthma, although not all patients have similar changes in each component of the remodeled airway.²²

Sputum, bronchoalveolar lavage, and bronchial mucosal biopsy specimens from elderly stable asthmatics have confirmed the presence of prominent eosinophilia, and CD4⁺ T lymphocytes, as seen with younger asthmatics.²³ Pathologic findings have been compared in young and older subjects who have died from fatal attacks of asthma.²⁴ Both old and young patients with asthma have thickened airway smooth muscle compared with normal subjects. Some studies have shown that older asthmatics with a longer duration of disease demonstrate increase in airway wall thickness. Another contributing factor to fixed airflow obstruction is a loss of elastic recoil. It has been reported in chronic asthma and it may be seen in varying age groups.^{25,26}

There is evidence that the airway function of young and middle age asthmatics may decline at a greater rate than normal subjects.^{27–29} The rate of decline increases with increasing age and in those who smoke cigarettes.^{28,30} These changes in asthmatics are variable because not all individuals show a steep rate of decline. The precise reasons for this individual variability have not been defined, although a history of atopy and marked bronchial hyperresponsiveness are 2 important risk factors for airflow obstruction.³¹ The long duration and severity of previous disease are also significant factors,³² although some elderly asthmatics with severe airflow obstruction report

having had a relatively short duration of symptoms, on the order of months to a few years. In asthmatics who develop asthma later in life, there is also evidence that lung function is reduced in some even before a diagnosis is made, declines rapidly shortly after diagnosis, and thereafter remains fairly stable.² In general, however, those with long-standing asthma have been shown to have more severe fixed airway obstruction.¹³ Airflow obstruction seen in the older asthmatic may not be attributable to asthma alone. The pathologic changes of asthma may synergize with those of normal aging, such as loss of lung elastic recoil, to affect both lung structure and function. The concept that aging and/or asthma duration can result in airway remodeling and cause “fixed” or irreversible airflow obstruction in this population can explain the lung function abnormalities seen in many older asthmatics. In a random survey of 1200 elderly asthmatics over aged 65 years from the Mayo Clinic, only 1 in 5 patients had normal pulmonary function even after administration of an inhaled short-acting bronchodilator.³³

CLINICAL FEATURES

The typical symptoms of asthma, namely, episodic wheezing, shortness of breath, and chest tightness, are the same in older asthmatics as in younger patients.³⁴ Often, the symptoms are worse at night and may be precipitated by exertion. However, compared with younger asthmatics, elderly asthmatics have greater morbidity¹¹ and lower scores on subjective assessments of health-related quality of life.³⁵ In addition to asthma symptoms, the elderly asthmatic is more likely to report poor general health, symptoms of depression, and limitation of activities of daily living.⁴

The symptoms of asthma in older adults are nonspecific, however, and are common to other conditions that are frequently seen with other comorbidities common in older adults. Dyspnea, for example, is a frequent complaint in those with heart disease, and paroxysmal nocturnal dyspnea can be due to both asthma and congestive heart failure. Many older patients limit their activity to avoid dyspnea; others may assume that their dyspnea is a result of aging and avoid seeking medical attention early in their disease course. Aging, per se, does not cause dyspnea. Cough is a prominent symptom and occasionally may be the only presenting symptom.³⁶ Wheezing, in contrast, may not be as prominent, and its presence is not specific and does not correlate with severity of obstruction.

Asthma is often triggered by environmental exposures. The Cardiovascular Health Study general population sample of asthmatics found that asthmalike symptoms were often brought on or made worse by exposure to the following factors: dust, smoke or fumes, and contact with animals, plants, or pollens.⁴ Also, similar to younger asthmatics, upper respiratory tract infections are common triggers of asthma symptoms and 1 community study showed that the majority of elderly patients who develop asthma after age 65 have their first asthma symptom preceded immediately by, or concomitant with, an upper respiratory tract infection.⁶

A history of atopy is a strong predictor of asthma in this age group, and current or previous allergic rhinitis, sinusitis, and nasal polyps are not uncommon. A comprehensive review of medications taken by patients may be helpful because aspirin, nonsteroidal antiinflammatory agents, and beta-blockers also can trigger symptoms. The physical examination in older patients who have asthma is usually nonspecific and a normal examination may obscure the diagnosis.

Studies have repeatedly shown that symptoms caused by asthma are frequently ignored or dismissed by older patients. Also, elderly patients, when compared with younger patients, have been shown to have a reduced perception of

bronchoconstriction.³⁷ This is true in both normal subjects and in asthmatics. This can delay medical intervention. Many older patients are fearful of having an illness and dying and are reluctant to admit that they have symptoms. Even when they do so, they may underestimate the symptoms, consider them a result of normal aging, or confuse these symptoms with those of other comorbid illnesses if present. Underreporting of symptoms in older adults may have many causes, including fear, depression, cognitive impairment, social isolation, denial, and poor medical literacy.

AIE may also be overlooked or misdiagnosed by physicians. Asthma symptoms are common to other conditions seen in older patients and asthma may be mimicked by these other diseases. Conditions such as congestive heart failure, emphysema and chronic bronchitis (COPD), chronic aspiration, gastroesophageal reflux disease (GERD), and endobronchial tumors can mimic asthma. Early morning wheezing is a prominent symptom of congestive heart failure. Asthma has not been found to be a risk factor for cardiovascular disease, unless there is a smoking history or a concomitant diagnosis of COPD.³⁸ Typical symptoms of GERD, such as vomiting and heartburn, may be absent in older adults. Reports of older patients with esophageal reflux proven by intraesophageal pH monitoring showed that chronic cough, hoarseness, and wheezing were present in the majority of patients studied.^{39,40} The relationship between GERD and asthma has been controversial. Attempts to control GERD with proton pump inhibitors (PPIs) has a limited impact on symptoms and lung function in patients with asthma and symptomatic GERD.⁴¹

OBJECTIVE MEASURES OF ASTHMA

Lung Function Testing

Objective measures of lung function with spirometry and peak flow measurements have been used to confirm the presence of asthma and monitor the disease. Airflow obstruction is usually present in symptomatic patients and reversibility can be shown after inhalation of a short-acting beta-agonist or anticholinergic agent. If complete reversibility of the airflow obstruction is seen, a diagnosis of asthma is more certain. COPD patients will also show airflow obstruction, but complete reversibility is not seen. Airflow obstruction is demonstrated by a reduction of the forced expiratory volume exhaled in 1 second (FEV₁) and the ratio of FEV₁/forced vital capacity (FVC). The Global Initiative for Chronic Obstructive Lung Disease (GOLD) COPD Guidelines use a fixed ratio of FEV₁/FVC of less than 70% to determine airflow obstruction.⁴² However, as aging occurs there is a loss of lung elastic recoil that reduces the FEV₁/FVC ratio in normal subjects. As a result, the diagnosis of airflow obstruction can be overestimated markedly. For example, in 1 study of patients over age 80, using the fixed ratio Turkeshi and colleagues⁴³ found that 27% of the subjects had airflow obstruction. It has been recommended that it is best to define airflow obstruction by the lowest 5% of the reference population (the lower limit of normal).^{44,45} Using this criterion to reevaluate the data, Turkeshi and colleagues⁴³ found airflow obstruction in only 9% of patients.

International standards regarding performance of spirometry have been published^{46,47} and widely accepted. Unfortunately, spirometry is underused in older patients.⁶ This contributes to the delay or failure to make a diagnosis. Although the effort-dependent maneuvers of spirometry may be difficult to perform in some patients because of physical and cognitive impairment, in 1 study a total of 585 elderly patients (81.8%) were able to perform spirometry according to accepted criteria.⁴⁸ Overall, geriatric patients are able to perform an imperfect, often unfinished, but acceptable forced expiration on this testing.⁴⁹ If airflow obstruction is found, attempts to demonstrate reversibility (postbronchodilator FEV₁ or FVC increases of >12% and 200 mL) should

be made.⁵⁰ Although reversibility testing becomes increasingly difficult with age, reliable data were found in a vast majority (94%) of subjects in 1 community study.⁵¹ A positive bronchodilator response makes a diagnosis of asthma is more secure; a negative response does not rule out asthma.¹¹ Unlike younger asthmatics, most elderly asthmatics show incomplete reversibility.¹² This can lead to a misdiagnosis of COPD.

Peak expiratory flow variability may be helpful in the diagnosis and follow-up of younger patients with asthma, but poor coordination and muscle weakness in some patients may lead to an inaccurate reading.^{52,53} A prospective study failed to demonstrate any advantage of peak flow monitoring over symptom monitoring as an asthma management strategy for older adults with moderate to severe asthma.⁵⁴

Airway obstruction may be absent at the time of testing and further evaluation may be needed to facilitate the diagnosis. Bronchoprovocation testing with a methacholine challenge can be useful and it is a safe, effective method to uncover asthma in older adults.^{55,56} A negative test rules out asthma; a positive test must be interpreted and include an assessment of pretest probability.⁵⁷

Use of Biomarkers

There are no particular biomarkers that distinguish younger and elderly asthmatics. AIE may be more neutrophilic,⁵⁸ but sputum neutrophil predominance is not specific to elderly asthmatics and can be found in older subjects without asthma as well,⁵⁹ and in younger asthmatics with severe disease.⁶⁰

Blood and sputum eosinophilia are common but not universal in asthmatics at any age. Older asthmatic subjects with evidence of atopy continue to exhibit sputum eosinophil numbers comparable to those seen in younger asthmatic subjects.⁶¹

In a study comparing older patients (>65 years) with fixed airflow obstruction and either nonsmoking asthmatics or COPD patients, those with asthma had significantly increased blood and sputum eosinophil counts and eosinophilic cationic protein levels.⁶² It seems, however, that aged eosinophils might have altered effector functions.⁶³ In a study examining age-related changes in eosinophil function, it was found that peripheral eosinophils from asthmatic subjects (age range, 55–80 years) exhibited decreased degranulation in response to chemotactic stimulation with IL-5 and lower superoxide production.⁶¹ Thus, eosinophils might be less functional in the elderly but continue to be associated with asthma.

Exhaled nitric oxide (FeNO) is used as an asthma biomarker in younger patients with asthma. FeNO is most accurately classified as a marker of T-helper cell type 2–mediated airway inflammation with a high positive and negative predictive value for identifying corticosteroid-responsive airway inflammation.⁶⁴ Higher levels have been found in very elderly asthmatics (over the age of 80 years), compared with a younger cohort of asthmatics (ages 18–30 years).⁶⁵ However, Columbo and colleagues⁶⁶ found that FeNO levels did not relate to disease activity measured by symptoms, lung function or exacerbations in a year-long study of 30 stable asthmatics ages 60 years and older. A Cochrane Database review concluded that the universal use of FeNO to help guide therapy in adults with asthma cannot be advocated. The main benefit of monitoring FeNO shown in this review was to help reduce exacerbations in a subgroup of patients who suffer from frequent exacerbations.⁶⁷ Overall, studies have suggested that FeNO likely has similar monitoring capabilities in both older and younger patients with asthma.

ASTHMA PHENOTYPES

There is increasing appreciation of the heterogeneity of asthma.⁶⁸ There are many phenotypic expressions of the disease that are defined by differences in clinical

presentations but give no insight into pathophysiologic mechanisms. A disease endotype is its functional or pathophysiologic mechanism. An appreciation of different disease endotypes among asthmatics has explained the differences in individual responses to various treatments and has also led to successful therapeutic trials of new biologic treatments. An appreciation of asthma phenotypes and endotypes in AIE has offered insights into the management of these patients.

Early-Onset Versus Late-Onset Asthma

The incidence of new-onset asthma is greatest in childhood and adolescence and, for many, asthma will persist into late adulthood. However asthma may develop at any age. When asthma occurs for the first time at an advanced age (>65 years) it has been labeled “late-onset asthma” and has been contrasted with “early-onset asthma” that often has been present for many decades and is more likely to have an atopic phenotype.^{12,13,69} Although these groups are indistinguishable by symptoms and medication requirements, the early-onset asthmatics are more likely to have more fixed airflow obstruction on spirometry¹³ and are more at risk for an acute exacerbation.⁷⁰

Atopic Asthma

The presence of atopy is common with asthma; more than 80% of children are allergen sensitized and this has proven to be a marker of more severe disease.⁷¹ A French study of asthma found only 14.7% of asthmatics over age 65 were sensitized to at least 1 antigen compared with 60.1% under age 21 years. Although there is a decreasing prevalence of atopy with age,^{72–74} a number of studies have confirmed allergic sensitization in certain populations of elderly asthmatics. An inner-city study by Rogers and colleagues⁷⁵ demonstrated that 60% of asthmatics greater than 65 years were sensitized to at least one antigen, the most common being cockroach.

Another by Busse and colleagues⁷⁶ showed that 41% of patients with moderate to severe asthma over the age of 60 were atopic, compared with 73% of patients between ages 18 and 35. Although most elderly asthmatics develop atopy at an earlier age, it has been documented that atopy can develop later in life. In the VA Normative Aging Study of older men (mean age, 61.2 ± 8.1 years), those with newly developed airway hyperresponsiveness were more likely to have developed sensitization to cats (23.9% vs 4.4%) compared with matched controls.⁷⁷

Obesity and Asthma

Obesity is a major risk factor for asthma.⁷⁸ The phenotypic features of the obese asthmatic are late-onset nonatopic asthma that is poorly controlled and often corticosteroid dependent. There is a predominance of female patients and often quality of life is poor and health care use high. Although obesity is known to adversely affect lung mechanics, it is also known to be a proinflammatory state.

The fact that weight reduction improves asthma control suggests involvement of inflammatory pathways secondary to obesity in the pathogenesis of asthma.⁷⁹ A Korean Longitudinal Study on Health and Aging (age >65 years) showed that the risk of asthma increased in proportion to the increase in body mass index in this elderly population. Additional studies specific to this age group are needed. Another phenotype of obesity and AIE occurs when a severe corticosteroid-dependent asthmatic remains on the corticosteroids for years and gains excessive weight as a complication of this treatment. There has been little research directed toward this problem.

Aspirin-Exacerbated Respiratory Disease

The association of aspirin and asthma was made by Samter and Beers in 1968 and was formerly called Samter triad.⁸⁰ The features of this syndrome include severe chronic rhinosinusitis and nasal polyposis, asthma and an exacerbation of severe asthma symptoms and rhinitis following the ingestion of cyclooxygenase-1 inhibitors such as aspirin and nonselective nonsteroidal antiinflammatory drugs. Unlike allergic asthma, this disease tends to develop in adulthood, will persist into later life, and occurs in patients without an atopic history. Among patients with nasal polyposis or chronic rhinosinusitis, the prevalence is about 10 and 9%, respectively. The majority of patients report a poor quality of life, chronic nasal symptoms, and a decreased sense of smell as important factors.⁸¹ It is now established that in these patients there is a dysregulation of proinflammatory and antiinflammatory lipid mediators: proinflammatory cysteinyl leukotrienes are markedly upregulated and the prostanoid prostaglandin E₂ is decreased.⁸² Corticosteroids and leukotriene pathway modifiers are the mainstays of therapy and aspirin desensitization has been used with success in some patients.

Asthma and Chronic Obstructive Pulmonary Disease Overlap Syndrome

COPD is an inflammatory disease of the airways that is caused mostly by cigarette smoke and combustion products of biomass fuels. Many asthmatics smoke cigarettes and likely have features of both conditions. These patients are thought to experience an overlap syndrome of asthma and COPD.⁸³ To date, when persistent airflow obstruction is present, there is no test that definitively distinguishes asthma from COPD. As a result, there is no universally accepted definition of the asthma-COPD overlap syndrome. A simplified practical approach to this definition “quandary” has been taken by the COPDGene investigators.^{84,85} The overlap syndrome is defined in patients with a diagnosis of COPD by GOLD criteria⁴² and asthma defined by a subjective report of a physician diagnosis of asthma before the age of 40. This definition avoids mislabeling COPD as asthma, because COPD would not likely be seen before age 40.

Other criteria for overlap syndrome of asthma and COPD that have been used include a history or evidence of atopy (eg, hay fever), an elevated total IgE, smoking greater than 10 pack-years, a postbronchodilator FEV₁ of less than 80% predicted and an FEV₁/FVC of less than 70%, and a 15% or greater increase in FEV₁ or a 12% or greater and 200 mL or greater increase after treatment with albuterol.^{86,87} When compared with patients with COPD alone,⁸⁴ the overlap patients have been described as a bit younger with less smoking intensity and with worse disease-related quality of life. They are more likely to have more severe and more frequent COPD exacerbations, and have a higher mortality.⁸⁸ Patients with overlap syndrome of asthma and COPD have been excluded from previous pharmacologic studies and further research is needed to identify the ideal therapeutic approach.

TREATMENT

The goals of successful asthma care have been established through global efforts⁸⁹ (Box 1). There are a number of barriers to reducing the burden of AIE, such as limited personal resources, inadequate access to medication, adverse environmental factors, significant comorbidities, and psychosocial challenges of aging. In addition, many elderly asthmatics show poor delivery technique, reducing the effectiveness of medication.^{90–92} However, most patients can be treated successfully with attention to

Box 1 Goals of asthma care	
1.	Limit symptoms of dyspnea, wheeze, chest tightness, and cough, day and night
2.	Provide normal daily activity level
3.	Maintain normal or near-normal lung function
4.	Reduce or eliminate asthma exacerbations; avoid emergency visits and hospitalizations
5.	Minimize use of rescue medication; use lowest dose and fewest medications possible
6.	Avoid side effects of medications

these cornerstones of asthma care: objective monitoring, avoidance of asthma triggers, pharmacotherapy, and patient education. These are outlined in [Table 1](#).

The monitoring tools discussed are most helpful in securing a diagnosis of asthma and comparing objective measures of lung function with the patient’s symptoms. Control of environmental influences is the second cornerstone of care. Both outdoor and indoor pollutants contribute to worsening asthma symptoms and new-onset asthma. Indoor pollutants include cooking and heating fuel exhaust, as well as insulating products, paints, and varnishes. The 2 main outdoor pollutants are industrial smog (sulfur dioxide particulate complex) and photochemical smog (ozone and nitrogen oxides). It is advisable to recommend that elderly asthma patients avoid, to the extent possible, exertion or exercise outside when levels of air pollution are high. Long-term passive cigarette smoke exposure has been linked to new-onset asthma in children and adults, as well as to the worsening of asthma symptoms, decreased lung function, and greater use of health services in those with preexisting asthma.^{93,94}

The third cornerstone of asthma care is pharmacologic therapy.^{89,95} Asthma is an episodic disease, the clinical presentation and natural history of which are highly variable from patient to patient and for any individual patient. Some patients have persistent symptoms and exacerbations from time to time. Others show long periods of remission, with sudden worsening with exposure to asthma triggers. Treatment protocols are based on this variability and use a step care pharmacologic approach based on the intensity of the asthma over time.^{89,95} As symptoms and lung function worsen, step-up or add-on therapy is given. As symptoms improve, therapy can be stepped down. Several composite control measures have been developed to help the clinician assess asthma control, including the simple 5-question Asthma Control Test.⁹⁶ The long-term goal of asthma treatment is directed at reducing airway inflammation. Medications referred to as “controller therapies” are used for this purpose. Inhaled

Table 1 Components of successful care for asthma in the elderly	
Monitoring	Use of self-assessment questionnaires, spirometry
Avoidance	Eliminate asthma “triggers,” such as allergen exposure and home/ workplace irritants
Treatment	Antiinflammatory therapy (inhaled corticosteroid) is the foundation of successful treatment for persistent asthma; short-acting bronchodilators for rescue therapy
Patient education	Provide the necessary tools for self-management including an action plan for exacerbation of symptoms

Table 2 Treatment choices for asthma in the elderly	
Therapy	Effectiveness/Risks
SABA	Used as rescue medication for acute symptoms. May reduce need for hospitalization. Effectiveness may be reduced in elderly. Tolerated well; caution with unstable cardiovascular disease.
SAMA	Effective bronchodilator; slower onset of action and fewer cardiac effects than SABA. Symptomatic urinary outlet obstruction, and closed-angle glaucoma uncommon, but potential risks.
ICS	Cornerstone of asthma care. Can reduce hospitalizations and mortality. Underutilized in elderly. Potential risks include decreased bone mineral density, increased fracture risk, and cataracts. May not be effective with neutrophilic-driven asthma
LABA	Effective long-acting bronchodilator; not to be used alone without ICS. Questionable cardiovascular risks; usually well-tolerated.
LAMA	Shown to be effective bronchodilator as add-on therapy to ICS and ICS/LAMA in the elderly asthmatic. Especially useful in patients with ACOS. Well tolerated in elderly.
LMA	Less effective agent in the elderly than with younger asthmatics, especially when given with ICS. Safe agent, any age
Anti-IgE	Given to atopic asthmatics not responding to ICS or LABA and/or LAMA. May be effective at advanced age.
Specific immunotherapy	May be effective in selected older atopic patients; risks and benefits must be weighed carefully

Abbreviations: ACOS, overlap syndrome of asthma and chronic obstructive pulmonary disease; ICS, inhaled corticosteroid; LABA, long-acting beta agonist; LAMA, long-acting muscarinic antagonist; LMA, leukotriene modifying agents; SABA, short-acting-beta agonist; SAMA, short-acting muscarinic antagonist.

corticosteroids have become the mainstay of controller therapy for persistent asthma symptoms. Commonly used medications are reviewed in [Table 2](#).

The last cornerstone of asthma management is the patient–clinician partnership. Patient education that fosters a partnership among the patient, his or her family, and those caring for the elderly patient is essential. Unfortunately it is often poorly implemented.⁹⁷ A multidimensional assessment of physical, psychological, cognitive, and social factors can impact successful treatment.⁹⁸ Self-management education provides patients with the skills necessary to control asthma and improve outcomes. The actions of the medications and potential side effects should be reviewed and the potential complications should be understood. Action plans should be written down and used as guidelines for daily care, including when to use oral corticosteroids, when to call the physician, and when to use emergency services.

SUMMARY

Asthma may occur at any age. In the elderly, it may be complicated by natural changes of the aging respiratory and immune systems. When asthma begins at a younger age, it is usually driven by atopic mechanisms and the disease may persist into old age. In some instances asthma may begin explosively at an advanced age; the pathogenesis is poorly understood. AIE is frequently associated with comorbidities of advancing age, such as heart disease, concomitant lung disease (eg, COPD), cognitive impairment, and depression. Unfortunately, geriatric-specific guidelines are not available

for the diagnosis and treatment of AIE. However, using a multidimensional assessment of physical, psychological, cognitive, and social factors can impact successful treatment.

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