

Near-Fatal Asthma in the Elderly

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Asthma affects the elderly as often as other age groups; however, it more often becomes fatal in the elderly. Unfortunately, asthma is often unmanaged or underdiagnosed in the older population. It is important for health care providers to recognize risk factors in the elderly and properly treat them before asthma becomes fatal. This article describes near-fatal asthma and identifies risk factors specifically for the elderly. Symptoms of asthma are reviewed as well as assessments and diagnostic tests to identify asthma severity and complications. Proper management needs to be urgently initiated to prevent worsening respiratory distress; this includes fast-acting drug treatments appropriate for elderly patients. Decompensated acute respiratory failure, secondary to severe asthma, requires the skills of an experienced anesthesiologist because these patients may rapidly deteriorate during induction and intubation. Ventilator management must include strategies to prevent worsening hyperinflation of the lungs. Elderly asthma patients have a higher mortality risk related to ventilator complications and other comorbidities.

Keywords: Asthma, Diagnosis, Elderly, Management, Treatment

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■ EPIDEMIOLOGY

One in 12 adults have asthma, and 9 people die of asthma every day.¹ The prevalence of asthma has increased by 15% in the past 10 years despite improvements in air quality and decrease in smoking statistics; this may be due to urbanization.¹ Most people think of asthma as a childhood disease. However, asthma is a common global disease affecting 300 million people, with high incidence occurring among all age groups including the elderly.² In a study by Tsai and colleagues,³ it was revealed that older patients are 5 times more likely to die of asthma than patients younger than 55 years, even after adjusting for comorbidities. The World Health Organization predicts that by the year 2050 the number of individuals older than 60 years will more

than double²; consequently, there will be an increase in asthma incidence among the elderly. Older individuals are often undiagnosed and live their life with restricted activities because of their shortness of breath, which they mistakenly attribute to normal aging.^{2,4} According to the Centers for Disease Control, adults 65 years or older were more likely to die of asthma than any other age group.¹

■ RISKS FOR FATAL ASTHMA

Comorbidities. Not surprisingly, comorbidities contribute to the increased mortality in elderly patients with asthma. The reported incidence of asthma in the elderly is most likely underreported because of coexistence or misdiagnosis with chronic obstructive pulmonary disease (COPD) or congestive

heart failure (CHF).⁵⁻⁷ Older patients are more vulnerable secondary to the effects of aging on deteriorating lung function, immunosuppression, muscle weakness, and cognitive decline.⁸⁻¹⁰ Multiple studies have shown that females are more prone to acquiring late-onset severe asthma after menopause. Severe asthma is also more prevalent with coexisting diagnoses of rhinitis, nasal polyps, and aspirin hypersensitivity.¹¹⁻¹⁴ Likewise, obesity contributes to systemic inflammation and compromises lung mechanics^{15,16}; it additionally contributes to the development of gastroesophageal reflux disease and obstructive sleep apnea, which can both exacerbate airway hyperresponsiveness (AHR) and hinder effective medical management. All these aforementioned comorbidities are interrelated and contribute to asthma exacerbation.^{11,17}

Drug-Induced Asthma. Comorbidities such as hypertension, CHF, glaucoma, and arthritis are common in older patients with asthma. Medications used to treat these diagnoses need to be prescribed with caution. Aspirin, β -blockers, angiotensin-converting-enzyme inhibitors, hormone replacement, and nonsteroidal anti-inflammatory drugs may cause exacerbation of asthma symptoms.⁴

Social Factors. Asthma control is directly influenced by psychosocial factors and instigating negative emotional responses such as anxiety, stress, or depression; these emotions may trigger a vagal response causing bronchial constriction.^{18,19} Elderly individuals often struggle with cognitive decline, polypharmacy, or difficulty affording their medicines, which may contribute to decreased medicine compliance and inadequate asthma treatment.⁶ Elderly patients have a higher tendency to smoke despite their respiratory problems; both smoking and exposure to secondhand smoke are common causes of increased asthma severity and mortality.^{6,20} In addition, elderly people who live in urban areas are exposed to higher levels of pollution. Those who have a low socioeconomic status tend to live with higher levels of indoor pollutants; in less developed countries, biomass fuels and wood burning are used for heating and cooking.⁶ In low-income housing, pet dander, cockroach, and mouse allergens; dust mites; mold; and lead paint combined with low ventilation contribute to increased allergen exposure.²⁰ Exposure to occupational allergens such as cleaning products, latex, and highly reactive chemicals has also contributed to late-onset severe asthma.¹¹

To make matters more complicated, older individuals have a decreased perception of their dyspnea and may remain undiagnosed for many years.^{6,9} This can be related to a misconception that shortness of breath or cough is normal for aging; in turn, seniors limit their daily activity. These symptoms may also be confused with CHF or COPD.⁷

Viral Infections. Fatal asthma attacks in the elderly are most often precipitated by viral respiratory tract infections; up to 56% present to the emergency department (ED) with respiratory infection.²¹ Common viral triggers leading

to hospital admissions are human rhinovirus and influenza A²¹; respiratory syncytial virus, picornavirus, and adenovirus contribute to a more severe asthma exacerbation.²² The viruses not only activate the innate immune cells along the epithelial lining of the lower respiratory tract, but they also have a direct effect on the airway smooth muscle (ASM) via engagement with Toll-like receptors (Toll-like receptor 3) presented by the ASM cells. The ASM then expresses various proinflammatory and anti-inflammatory cytokines (interleukin 5 [IL-5], IL-6, IL-8, IL-11, and IL-1 β) that induce constriction and decrease sensitivity to steroids and β_2 -agonist bronchodilators, the 2 main treatments for asthma.²³ The viral infection causes epithelial damage and exposure of the basement membranes, which can take months to completely heal, increasing the potential for a secondary respiratory infection. The most common offenders are *Haemophilus influenzae*, *Streptococcus pneumoniae*, and gram-negative bacteria.^{12,24} Additional studies show that positive serology for *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* may contribute to asthma chronicity and severity.^{9,12} With an already primed immune system and neutrophilia, a second infection can cause overwhelming systemic inflammation and oxidative stress, which can be fatal in an already compromised elderly patient.

Inflammaging. Asthma studies have shown that elderly patients are more prone to a severe AHR and a weakened immune response.^{6,25} This is the consequence of a coexisting chronic low-grade inflammation that occurs in an aging immune system called immunosenescence or “inflammaging.”^{26,27} Both obesity and old age contribute to decreased asthma control secondary to the related chronic inflammatory state that is induced by poor perfusion to adipocytes and up-regulation of proinflammatory cytokines.^{16,26} This in turn causes decreased effectiveness of the airway macrophages to engulf inflammatory cells and decrease airway inflammation.

■ CLASSIFICATION OF SEVERE ASTHMA

A thorough medical history will help to predict those at risk for severe or fatal exacerbations. Any history of prior intubation or a severe asthma exacerbation requiring hospital admission in the past 5 years or more than 1 visit to the ED in the past year would be considered a red flag.⁹ Exacerbations can be unpredictable, triggered by a viral infection or allergen, and last anywhere from minutes to days. The severity of an exacerbation can vary from mild to severe; however, the risk for a fatal asthma attack is measured by the frequency of severe exacerbations. Persons with asthma who have utilized 2 or more full canisters of inhaled short-acting β_2 -agonists (SABAs) per month or needed more than 1 course of oral corticosteroid treatment within the past year would also be considered at higher risk.⁹

Severe asthma is also assessed by a history of symptom frequency over the previous month. Persistent severe asthma is defined by symptoms that occur several times throughout the day with nightly awakenings, SABA use several times daily for symptom control, and significant limitation in activities of daily living.⁹

■ PULMONARY FUNCTION TESTS

Peak Expiratory Flow. Pulmonary function testing is crucial in generating a proper diagnosis of asthma. Peak expiratory flow (PEF) is an easy test done in the ED. It is an assessment of airflow obstruction and measured during a maximally forceful and rapid exhalation. Initial ED assessment should include PEF and a repeat measurement after the first hour done to monitor treatment effectiveness. Normal is considered greater than 80% of an expected value calculated by sex, age, and height. Less than 40% expected value, or 200 L/min, would be considered severe obstruction, and less than 25% is life threatening.⁹ Unfortunately, PEF is not useful for diagnosis of asthma, and without a baseline measurement, it is not very reliable. During a severe exacerbation, most patients are unable to perform this test. Nonetheless, in severe asthma and in individuals with difficulty perceiving the severity of asthma, PEF is a good monitoring tool that can be administered daily and as needed at home.

Spirometry. Spirometry provides a measure of flow and obstruction; however, measurements are not very accurate if the patient is unable to coordinate breathing as directed. It is also underused in the older population.⁷ It is well known that the natural process of aging deteriorates organ function as well as decreasing lung compliance and lung capacity; therefore, pulmonary function testing results in older individuals may resemble COPD. The main difference between asthma and COPD is the reversibility of airflow obstruction. McDonald et al describe in depth the characteristics of asthma-COPD overlap, which occurs often in adults older than 65 years.²⁷

Forced Expiratory Volume in 1 Second and Forced Vital Capacity. Obstruction is first measured by the forced expiratory volume in 1 second (FEV₁). This measurement is similar to the PEF with normal values calculated by the person's age, sex, height, and race. An FEV₁ between 80% and 120% of predicted value is considered normal. Severe asthma is defined as less than 60% predicted,⁸ and less than 30% is considered life threatening.²¹ Along with an FEV₁, the forced vital capacity (FVC), or the total volume of exhaled air, is measured; in patients with asthma and elderly patients, it would be below predicted value.⁵

The ratio of FEV₁ to FVC is a more accurate measure of obstruction because a lower proportion of FVC is expired in the first second; this is defined as a value of less than 70%. Elderly patients may have a normal decrease in lung function with an FEV₁/FVC ratio as low as 65%; therefore, it

is recommended that results be compared with the lower limit of normal values for a comparative population of the same age, height, and gender.^{9,28} Some elderly patients may be unable to comply with the breathing maneuvers required for assessment.⁴

Bronchodilator Challenge. Most significant in diagnosing asthma is the assessment of the reversibility of obstruction. Obstruction is reversible if there is greater than 12% increase in the FEV₁ after bronchodilator challenge, keeping in mind that elderly persons require a longer time to achieve full bronchodilator effect than do younger patients with asthma.⁴ Also, older patients with asthma may respond better with a combination of albuterol and ipratropium bromide.⁴ Unfortunately, this test cannot be done during hospital admission for asthma because it requires withholding all treatment prior to the examination. Careful administration of a methacholine challenge test is done to measure AHR in patients with asthma with normal pulmonary function, which occurs more often in elderly patients with late-onset asthma; however, older patients may require only half the standard dose.⁴

■ ASSESSMENT AND TESTING

Presentation. On presentation, the patient may appear anxious or agitated while sitting in a tripod or upright position and using accessory muscles to breathe. Wheezing is usually audible, and the patient may be diaphoretic, tachypneic, and tachycardic. During a severe episode, the patient will have difficulty speaking in single words; therefore, answering history questions will be difficult. A brief history should focus on aforementioned risks for fatal asthma and onset, duration, and trigger factors for asthma symptoms. Any change in mental status, decrease in wheezing, bradypnea, bradycardia, or cyanosis could indicate worsening hypercapnia and respiratory arrest; this indicates a need for intubation.²⁹ Keep in mind differential diagnoses that may have similar presentations (Table).

Pulsus Paradoxus. Pulsus paradoxus (PP) is a well-known phenomenon that may occur with severe asthma.²¹ A decrease in systolic blood pressure of greater than 10 mm Hg during each inspiration can be easily measured in the arterial waveform. Other methods are more difficult to assess, such as the use of cuff sphygmomanometry and palpation; however, with practice, these assessment skills can become invaluable. Pulsus paradoxus is caused by the increased intrathoracic pressure from use of accessory muscles and hyperinflation of the lungs. Even a small improvement in airway caliber or worsening respiratory muscle fatigue can decrease the presence of PP.³⁰

Hyperinflation. Airway hyperresponsiveness occurs both in the main bronchi and throughout the terminal bronchioles that have increased proliferation of ASM as well as decreased elastic recoil.¹² The smaller airways are

TABLE Differential Diagnoses

Cardiac
Congestive heart failure
Myocardial infarction
Valvular disease
Psychiatric
Hyperventilation disorder
Respiratory:
Anaphylactic reaction
Allergic aspergillosis
Aspiration
Bronchitis
Chronic obstructive pulmonary disease
Foreign body
Interstitial lung disease
Laryngeal edema
Neoplasm
Pneumonia
Pulmonary edema
Pulmonary embolus
Vocal cord dysfunction

more susceptible to mucus plugging and severe narrowing causing air trapping; this contributes to airflow limitation. During a severe exacerbation, tachypnea and increased tidal volume occur to increase ventilation; however, increased airway resistance and insufficient expiratory time prevent the lungs from fully exhaling. Recruitment of accessory muscles increases intrapleural pressure, causing “auto-PEEP” (positive end-expiratory pressure), and contributes to alveolar collapse and increased dead space ventilation.³¹

Increased intrathoracic pressure contributes to additional complications; it progressively increases the work of breathing by shortening the diaphragmatic length and minimizing its ability to generate optimal expiratory force.³² In addition, the increased intrapleural pressure decreases preload, compresses the pulmonary capillaries, and increases afterload, consequently decreasing cardiac output; this is the cause of PP.³¹ In elderly patients with underlying heart disease, increased intrathoracic pressure can be fatal; older lungs are more susceptible to rupture and pneumothorax.

Chest X-ray. If pneumothorax is suspected, then a chest x-ray (CXR) would be warranted. In less severe asthma cases, a CXR would not be needed; however, in the elderly, coexisting CHF and COPD are more likely to contribute to complications. In addition, if an infectious process is suspected, a CXR would be needed to rule out pneumonia.

Cardiac Monitoring. All elderly patients with a history of heart disease should be continuously monitored. Elderly patients are more likely to develop atrial fibrillation or arrhythmias. An electrocardiogram should also be ordered to rule out myocardial infarction or CHF. Airflow obstruction may induce a right ventricular strain pattern on electrocardiogram.³⁰

Laboratory Tests. Laboratory test results do not provide much direction for asthma management; however, in the elderly patient, a BNP (B-type natriuretic peptide) would be helpful in ruling out heart failure by detecting ventricular stretch factors. Leukocytosis is common, but not a diagnostic indicator of infection; furthermore, recent use of corticosteroid or epinephrine would contribute to demargination of neutrophils, increasing total white blood cell count.³⁰ In critically ill elderly patients, electrolytes need to be monitored. Multiple β -agonist treatments may decrease serum potassium, magnesium, and phosphorus, especially when diuretics are needed. In sicker patients, respiratory failure and excess catecholamines contribute to a rise in lactate.²¹

Recent studies have been focusing on searching for monitoring tools to manage severe asthma. According to the International European Respiratory Society/American Thoracic Society, serum immunoglobulin E can be useful in identifying immunoglobulin E-dependent allergic asthma; however, use of sputum eosinophil counts and exhaled nitric oxide is not recommended for use by clinicians outside specialty centers for research and asthma.¹²

Arterial Blood Gases. An initial arterial blood gas (ABG) is not very useful in predicting asthma severity. Monitoring clinical signs of improvement is more reliable. The trending of ABG results is best explained by the “Fish Diagram” illustrated by Dr Crocco (Figure).³³ At the onset of the asthma attack, the patient’s increased respiratory rate will “blow off” CO_2 ; therefore, ABG results will show a decreased PaCO_2 and a correlating increase in pH.³⁰ As the exacerbation worsens, and the patient is no longer able to keep up with the ventilatory demand, fatigue sets in, and CO_2 is retained. At this point, the PaCO_2 and pH may look normalized despite the patient’s worsening condition and progressing respiratory failure, acidosis, and hypercapnia.³³ A “normalized” ABG may be an ominous sign when clinically the patient is not improving, and impending respiratory failure can occur before hypercapnia.^{21,34} Respiratory compromise can be well monitored with capnography, a measure of expiratory concentration of CO_2 , which correlates well with PaCO_2 .³⁰

■ EMERGENCY TREATMENTS FOR FATAL ASTHMA

Oxygen. Immediate high-concentration oxygen should be administered via face mask and oxygen saturation closely monitored. Start with high concentration, FIO_2 100%. Oxygen

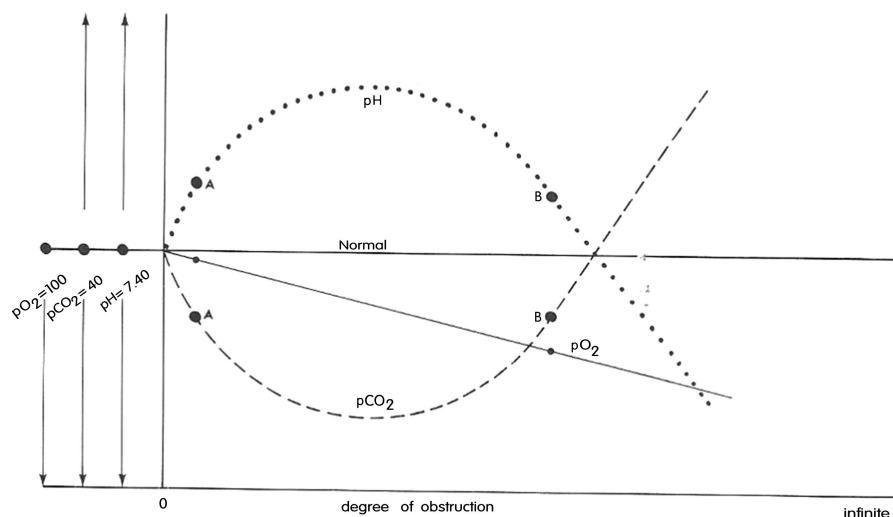


Figure. Fish Diagram by John A. Crocco, MD. A, Onset of asthma attack. B, Progression to respiratory failure. Reprinted with permission from Dr Crocco.

can then be weaned to maintain saturation greater than 93% for elderly persons.³⁵

β -Agonists. A continuously inhaled SABA such as albuterol should be initiated immediately at a dose of 10 to 15 mg over 1 hour.^{21,35} Short-acting β_2 -agonists are the most effective fast-acting bronchodilators that relax ASMs, but they have no effect on airway inflammation.³⁶ Of note, studies have revealed that elderly patients have less β_2 -adrenergic receptors on their ASMs, and their response to SABAs may be decreased.⁷ In addition, the elderly may experience increased adverse effects from β -agonists, such as tremors, tachycardia, hypokalemia, and leg cramping.⁶ There is currently no evidence to support the use of IV β_2 -agonists for treatment of asthma.³⁵ SABAs should be utilized only as a rescue medicine, not for long term management. Asthma symptoms requiring SABA use more than 3 times a week indicate uncontrolled asthma.⁹

Long-acting β -agonists (LABAs), such as salmeterol, should not be used in urgent care.⁶ Although SABAs have been shown to be less effective in the elderly, long-term management with LABA is just as effective as in the younger population and has decreased mortality in the elderly.^{6,7} Monotherapy with inhaled β -agonists is associated with increased mortality rates; therefore, it is recommended that inhaled corticosteroids be given in conjunction with LABA to decrease airway inflammation.^{12,36}

Anticholinergics. Ipratropium bromide is a muscarinic receptor antagonist that promotes bronchodilation and decreases mucus secretion. Elderly patients with decreased response to albuterol achieve a better response with combined administration of albuterol and ipratropium^{20,30}; however, it is not recommended for mild asthma.³⁵ A dose of ipratropium 0.5 mg every 20 minutes times 3 doses may be mixed with albuterol.

Epinephrine. Older age and heart disease are contraindications for use of epinephrine because it may cause hypertension, tachycardia, and myocardial ischemia. According to the 2014 Global Initiative for Asthma report, epinephrine should be reserved for treatment of anaphylactic asthma and angioedema.³⁵ A subcutaneous injection of 0.3 mg may be given every 20 minutes up to 3 doses as needed for treatment of anaphylaxis.

Corticosteroids. Early administration of 125 mg IV methylprednisolone will help to decrease airway inflammation by reducing eosinophils and inflammatory cells and also decreasing transcription factors for proinflammatory cytokines.³⁶ Unfortunately, effectiveness of corticosteroids may take more than 2 hours, and evidence shows that oral corticosteroids are just as effective as IV.³⁵ There may also be some added benefit from conjunctive administration of inhaled corticosteroids.³⁵

Magnesium. Magnesium relaxes the ASM by preventing the influx of calcium into the cells; it also decreases neutrophil-mediated inflammation.³⁷ If the patient does not show clinical signs of improvement, then 2 g IV magnesium is given over 20 minutes, which has been shown to be beneficial for treatment of severe asthma.³⁵ Alternatively, it can be administered via nebulizer in conjunction with β -agonists. Usually there are minimal adverse effects; however, rapid infusion has been known to cause hypotension and hypermagnesemia, which can also cause muscle weakness.³⁴

RESPIRATORY FAILURE

Signs of deterioration, such as decreased wheezing or silent chest, alteration of mental status, hypoxemia, bradypnea, or fatigue of accessory muscles, indicate a need for mechanical ventilation. Temporary mechanical ventilation may be emergently needed to allow more time for bronchodilators and

anti-inflammatory treatments to take effect. An experienced clinician should proceed with this high-risk rapid-sequence intubation. Postinduction hypotension, arrhythmias, and rapid desaturation should be anticipated with available vasopressors, atropine, and IV fluids. Asthmatics are also at high risk for laryngospasm and bronchospasm during intubation procedures.²¹

Induction. To start, pretreatment with 1.5 mg/kg of IV lidocaine for a good 3 minutes prior to induction will help to minimize bronchospasm of the upper airways.³⁰ Second, the patient needs to be preoxygenated manually at a low respiratory rate to prevent further overinflation of the lungs and consequent hypotension. Etomidate (0.3 mg/kg) is the primary drug of choice for induction sedation; however, ketamine (1-2 mg/kg) may be a better choice in patients with severe asthma.^{30,34} Ketamine has a catecholamine effect and causes bronchodilation without respiratory depression.³⁷ Choice agents for induction paralysis are succinylcholine (1.5 mg/kg) or rocuronium (1 mg/kg).^{21,30,34} Postintubation sedation will be critically necessary to prevent ventilator dyssynchrony and help decrease hypercapnia. Propofol is a good sedative agent for asthma because of its bronchodilating effects; morphine, however, has a histamine effect and should be avoided.^{21,30,37}

Ventilator Goals. Ventilator management of an elderly patient with severe asthma can become challenging. The goal is to prevent hyperinflation of the lungs and monitor for complications of hyperinflation. Initial ventilator settings should focus on low tidal volume (6 mL/kg) and respiratory rate (10-12 breaths/min), as well as longer expiratory time (>1:3).^{21,30,34} This will help prevent an increase in already present intrinsic PEEP. Initial extrinsic PEEP should be set at 0 to 5 cm H₂O, and FIO₂ should be weaned to maintain goal oxygen saturation.²¹

To prevent hyperinflation, the plateau pressures need to be closely monitored and maintained below 30 cm H₂O.^{21,29,34} Monitoring ventilator flow waveforms for air trapping is also helpful. Serial ABGs or venous blood gases are drawn to monitor serum pH, and continuous capnography provides a good monitor for hypercapnia. The goal pH is more than 7.2 (some studies allow 7.15); this is called permissive hypercapnia. This is a well-studied strategy to prevent barotrauma by allowing hypoventilation and subsequent hypercapnia to rise gradually. Drastic changes in pH can cause hemodynamic compromise. As long as the pH falls slowly and later has a gradual return to baseline, permissive hypercapnia is well tolerated.^{21,29,30}

CONCLUSION

Severe and fatal asthma is usually preventable. The problem is that it is often not properly diagnosed and therefore uncontrolled. Underdiagnosis occurs both from the elderly patient's misconception of "growing old" and from care

providers not recognizing that asthma occurs in more than 11% of the elderly population.¹ It is vital that clinicians and nursing staff be familiar with presenting symptoms of severe asthma and rapidly manage these patients appropriately before respiratory failure sets in. Once diagnosed, risk factors such as smoking and obesity can be addressed, and proper medical management can drastically better quality of life for these older individuals. Most importantly, it is essential to institute an asthma action plan to prevent frequent visits to the ED and treat exacerbations before they turn fatal.

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