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Case Review

A 56-Year-Old Man With an Asthma Exacerbation

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A local flight team was activated for an interfacility transfer of a 56-year-old man with a history of asthma from a local community hospital to a tertiary care center for respiratory failure. The patient had a history of severe asthma in the past that had been not been well controlled. He had been admitted multiple times to this particular local hospital in the past and had required intubation on several of those occasions. On this particular date, he had been admitted with increased work of breathing and dyspnea that had progressed since the night before. He reported that during the last several days he had a cough and concurrent typical upper respiratory symptoms necessitating the use of his daily albuterol inhaler. Once in the hospital, staff noted that his respiratory condition worsened despite aggressive albuterol therapy, corticosteroids, and magnesium sulfate. Thus, an arterial blood gas test was completed, which immediately showed a severe respiratory and concomitant metabolic acidosis with a pH less than 7.0 (Table 1). Because of the imminent respiratory failure, the patient was subsequently intubated for increasing hypoxia as well as respiratory failure by the staff at the sending hospital before the flight team's arrival. He was intubated with standard doses of ketamine and succinylcholine, and sedation was continued with intravenous propofol.

While the crew was en route to the scene, a variety of ventilation modalities were attempted without significant success. The patient remained tachypneic and hypoxic and had elevated plateau pressures that were greater than 30 mm Hg. In fact, the sending physician became so concerned about the patient's acidosis that she initiated a sodium bicarbonate bolus and drip before the team's arrival. The etiology of the respiratory compromise was identified as a right middle and lower lobe pneumonia that was treated with intravenous vancomycin and piperacillin/tazobactam. Basic laboratory profiles were completed and were only significant for leukopenia of 1.3. There was no history of altered white blood cell counts in the past. Additionally, an electrocardiogram was completed and showed a sinus tachycardia without evidence of myocardial ischemia.

Upon arrival, the team found the patient intubated and sedated in the semi-Fowler's position with intravenous propofol infusion to maintain a modified Ramsay score of 4 to 5. Initial ventilator settings per the sending facility were volume control at a rate of 28 breaths per minute with a tidal volume of 550 mL, a positive end-expiratory pressure (PEEP) of 5 cmH_2O, and a fraction of inspired oxygen of 100%. (The patient's ideal body weight was 73 kg.) The patient was noted to have the following set of vitals at the time of patient contact: pulse of 142, blood pressure of 81/63 mm Hg, spontaneous respiratory rate of 24 breaths per minute, end-tidal carbon dioxide of 24, and oxygen saturation of 98%.

Once the patient was evaluated, the team quickly attempted to provide an extended expiratory phase because the patient was spontaneously triggering his own faster intrinsic rate. Thus, the crew lowered the inspiratory phase to 0.7 seconds and made the ventilator rate 10 breaths per minute. During the course of the relatively short flight, sedation was continued with propofol and fentanyl. However, during the 8 minutes when the patient was in transit, no significant gains were made in the patient's overall status. Just before landing at the accepting facility, the patient became acutely hypotensive with systolic blood pressures in the 70s and 80s. Aggressive fluid resuscitation was initiated.

Table 1
The initial Arterial Blood Gas of the 56-year-old Man With Respiratory Failure

Components of the Arterial Blood Gas	Values
pH	6.96
PCO_2	94
PO_2	81
HCO_3	22

Once at the arriving center, the patient was transferred to the intensive care unit. His respiratory status remained essentially unchanged. Using a low volume strategy with a prolonged expiratory phase, the pulmonary staff was able to oxygenate him marginally. However, it was at this point that his overwhelming sepsis declared itself, and despite aggressive fluid therapy, he required the addition of multiple vasopressors to support his hemodynamics. During the course of the next 24 hours, his status continued to deteriorate. Ultimately, he required the use of 3 agents to support his blood pressure including dopamine, norepinephrine, and vasopressin. He underwent an echocardiogram to evaluate for additional etiologies of his hypotension. He was noted to have global dysfunction of his myocardium with an overall ejection fraction of 10%. Repeat chest x-rays were completed and showed worsening of his right upper and middle lobe pneumonias. Throughout his hospitalization, he remained bronchospastic despite all the aggressive therapies. In the end, his family made the decision to withdraw care because of his poor overall condition and prognosis. He was terminally extubated and passed soon after with the diagnosis of acute asthma, pneumonia, and septic shock.

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Discussion

Epidemiology

Asthma is a routinely encountered diagnosis that staff members working in emergency departments (EDs) routinely encounter. It accounts for more than 2 million visits to EDs annually with approximately 4,000 deaths in the United States.[1] The management of asthma is a significant burden to the US health care budget with annual costs exceeding $56 billion.[1] Luckily, the majority of these patients do not need hospitalization. In the United States, approximately 450,000 patients are hospitalized for their respiratory distress, and of these only 4% are admitted to an intensive care setting.[2] Deaths related to asthma occur as a result of cardiopulmonary arrest, cerebral anoxia, and iatrogenic treatments (ie, barotrauma and ventilator-associated pneumonias) and are highest in the elderly (age greater than 65 years), women, and those from disadvantaged backgrounds where their asthma is poorly controlled.[3]

For those patients with the predilection of near-fatal asthma (NFA), there are a variety of factors that can predispose patients to severe disease. By definition, asthma is an allergic disease that is characterized by bronchial hyperresponsiveness, increased vascular permeability, and spasms of bronchial smooth muscle. Subsequently, the simultaneous release of vascular mediators results in clinical signs and symptoms including wheezing, dyspnea, chest tightness, and coughing. Thus, when patients are exposed to certain precipitants, exacerbations occur. Factors that can induce asthma exacerbations can include viral upper respiratory infections, nonsteroidal anti-inflammatory drugs, tobacco use, stress, and illicit drug inhalation (crack cocaine and heroin).[3,4] As in the case described, between 12% and 56% of ED presentations of acute asthma exacerbations occur as a result of viral or bacterial upper respiratory infections including influenza A and rhinoviruses.[4]

Signs and Symptoms

In cases of severe asthma exacerbations, there are 2 distinct patterns of presentation. The most common (80%-85%) occurs over the period of several days with worsening dyspnea, bronchospasm, chest tightness, and cough. The indolent nature of the exacerbation is caused by increased bronchial secretions (ie, mucous plugging) and eosinophilic inflammation. The less common group of patients (15%-20%) has an acute respiratory distress response (acute asphyxic asthma) that occurs in a short time in response to a specific irritant.[2]

When diagnosing patients with suspected acute asthma exacerbation, it is imperative to not only exclude other mimics but also to identify concurrent diagnoses. There are a variety of respiratory pathologies that can present with acute bronchospasm and respiratory distress including congestive heart failure, chronic obstructive pulmonary disease, anaphylaxis, upper airway abstractions, and pulmonary embolism among others (Table 2).

Table 2
Common Diagnostic Mimics of Acute Asthma[1,2,5,6]

Congestive Heart Failure	Croup
Coronary artery disease	Bronchiolitis
Chronic obstructive pulmonary disease	vocal cord dysfunction
Pulmonary embolism	Viral upper respiratory infection
Vocal cord dysfunction	Hyperventilation syndrome
Pneumonia	Tracheomalacia
Foreign body aspiration	Bronchitis

Table 3
Risk Factors for Increased Asthma Morbidity and Mortality[1,2,5]

Previous Asthma Exacerbations	Illicit Drug Use
Multiple annual emergency department visits or inpatient hospitalizations	Concurrent tobacco use
Heavy use of beta-agonist therapy	Multiple comorbidities
Low socioeconomic status	Psychiatric disease

Once the diagnosis of asthma is made, providers must risk stratify the patient to determine treatment modalities and final disposition based on objective clinical signs and relevant history. In patients with a history of asthma in the past, it is imperative to identify risk factors and important history to determine the best management approach. Patients with status asthmaticus (SA) and NFA present with common signs and symptoms previously described in this article and in general medical education for asthma (ie, bronchospasm, dyspnea, coughing, and so on). However, the key difference is that in these cases patients do not respond to typical initial treatments and progress to respiratory failure. In this population, the mortality increases significantly to between 10% and 25%.[2]

While completing the initial assessment, risk factors for SA and NFA must be identified and can include a history of severe exacerbations, multiple hospitalizations, and heavy beta-agonist use (Table 3). The provider then can risk stratify the patient based on the severity of illness to determine the level of care needed during the initial hospitalization (Table 4).

Treatment

The treatment of severe asthma exacerbations focuses primarily on the prevention of the progression of disease. Initial therapies for all asthma-related complaints include the use of supplemental oxygen and beta-agonist therapy. Minimal to moderate hypoxemia is a common finding in patients with the disease; however, patients with a $PaO_2 < 55$ mm Hg are quite rare.[3] Supplemental oxygen will work to mitigate the V/Q mismatch that occurs secondary to obstruction and bronchoconstriction. Therefore, the number of patients exhibiting air hunger will be decreased. In most cases, a fraction of inspired oxygen of 30% to 50% will suffice and will not cause complications such as respiratory depression or hypercarbia.[3,5]

Concurrent to the administration of oxygen is the use of short-acting beta-agonists such as albuterol, levalbuterol, and in some cases pirbuterol. This class of medications relieves signs and symptoms by relaxing smooth muscle, decreasing bronchoconstriction, and decreasing vascular permeability. The medication can be given using a metered dose inhaler with a spacer or nebulization with equal efficacy. Typical dosing for the use of a metered dose inhaler is 4 to 8 puffs every 20 minutes and 2.5 to 5.0 mg inhaled via a nebulizer, respectively.[2] However, it is important to note that there is a significant group of “nonresponders” to beta-agonists who exhibit minimal relief to inhaled therapy. In these cases, other adjunct therapies must be considered including parenteral therapy as noted later. In critically ill patients, continuous inhaled albuterol therapy can be used for total administration for up to 15 mg.[2] Additionally, the use of terbutaline or epinephrine has also been identified as a way to parenterally provide relief with beta-agonist therapy. Epinephrine is often an overlooked form of therapy but can also produce unwanted cardiovascular effects including tachycardia and hypertension because of its concurrent alpha 1 effects. Despite these warnings about the use of epinephrine, it is routinely used with minimal complications. Studies in the past have shown that concerns about its adverse effects have not been identified, but prospective random trials