Managing a severe acute asthma exacerbation

Act fast to prevent respiratory collapse

By Jin Xiong Lian, RN

Caring for a patient with an acute asthma exacerbation or status asthmaticus is quite common in critical care. In this article, I’ll review the pathophysiology and management of a severe asthma exacerbation so you’ll be prepared to monitor your patient’s response to therapy and intervene appropriately should the patient’s clinical status deteriorate rapidly.

A chronic inflammation

Asthma is a chronic inflammatory disorder of the airway characterized by airway hyperresponsiveness, mucus hypersecretion, and reversible airflow limitation. Chronic inflammation associated with asthma thickens the airway walls or changes their structure, a process known as airway remodeling. In an acute episode, the patient also has airway edema and bronchoconstriction. Asthma is variable in terms of genotypes, clinical phenotypes, susceptibility to environmental factors, pathogenesis, expression, severity, and response to treatments.1-3

Asthma triggers include viral respiratory infection, exposure to an allergen or irritant, exercise, stress, smoking, cold air, gastroesophageal reflux, nonadherence to pharmacologic therapies or sudden withdrawal from corticosteroids, and consumption of certain foods or intake of certain drugs such as aspirin, beta-blockers, nonsteroidal anti-inflammatory drugs, cocaine, or heroin.1,2,4-6

Asthma exacerbations can be classified as mild, moderate, severe, or respiratory arrest imminent.1 [For more details, see Severity of asthma exacerbations.] A severe exacerbation, the focus of this article, is characterized by severe airflow limitation and dynamic hyperinflation (also called air trapping), in which end-expiratory lung volume increases because patients can’t exhale fully, but are trying to maintain near-normal gas exchange.2,4-8 Bronchospasm, edema,
asthma exacerbation
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Excessive secretions, and mucus plugs lead to progressive airway narrowing, increased airway resistance, insufficient expiratory time, and respiratory muscle fatigue. As a result, dynamic hyperinflation and elevated intrinsic positive end-expiratory pressure (PEEPi) occur. In a healthy patient, a small amount of air remains in the lungs at end-expiration, creating normal PEEP of 3 to 5 cm H₂O. However, in a patient with dynamic hyperinflation secondary to an asthma exacerbation, more air is left in the lungs at end-expiration than the physiologic normal volume, resulting in increased PEEPi.

Airflow obstruction diminishes ventilation, reduces oxygen intake, and increases ventilation/perfusion (V/Q) mismatching. In addition, frequent coughing to clear sputum or airway secretions, using accessory muscles of respiration, and being agitated increase oxygen consumption and carbon dioxide (CO₂) production. Without timely and appropriate treatment, a patient with an acute severe asthma exacerbation will develop hypoxemia and hypercapnia, leading to anaerobic metabolism, lactic acidosis, and acid-base imbalance.

Clinical manifestations of an acute asthma exacerbation vary among patients and by the severity of asthma. The common signs and symptoms include cough, wheezing, shortness of breath, chest tightness, diaphoresis, tachypnea, and tachycardia. Remember that the absence of typical signs and symptoms doesn’t mean that the patient’s condition isn’t getting worse.

### Severity of asthma exacerbations

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Respiratory arrest imminent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>Patient can walk, lie down, and talk in sentences</td>
<td>Patient prefers sitting, talks in phrases</td>
<td>Patient is leaning forward and can only say a few words between breaths</td>
<td>Drowsy or confused</td>
</tr>
<tr>
<td>Alertness</td>
<td>May be agitated</td>
<td>Usually agitated</td>
<td>Usually agitated</td>
<td>Paradoxical thoraco-abdominal movement</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Increased</td>
<td>Increased</td>
<td>Often greater than 30 breaths per minute</td>
<td></td>
</tr>
<tr>
<td>Use of accessory muscles of respiration</td>
<td>Usually not</td>
<td>Usually</td>
<td>Usually</td>
<td>Paradoxical thoraco-abdominal movement</td>
</tr>
<tr>
<td>Wheezing</td>
<td>Moderate, and often only at end-expiration</td>
<td>Loud</td>
<td>Usually loud</td>
<td>Absent</td>
</tr>
<tr>
<td>Pulse</td>
<td>Less than 100/minute</td>
<td>100-120</td>
<td>Greater than 120</td>
<td>Bradycardia</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>Absent (less than 10 mm Hg difference)</td>
<td>May be present (10 to 25 mm Hg difference)</td>
<td>Often present (greater than 25 mm Hg difference)</td>
<td>Absent, suggesting respiratory muscle fatigue</td>
</tr>
<tr>
<td>PaO₂</td>
<td>Normal</td>
<td>Greater than 60 mm Hg</td>
<td>Less than 60 mm Hg</td>
<td></td>
</tr>
<tr>
<td>PaCO₂</td>
<td>Less than 45 mm Hg</td>
<td>Less than 45 mm Hg</td>
<td>Greater than 45 mm Hg</td>
<td></td>
</tr>
<tr>
<td>SaO₂</td>
<td>Greater than 95%</td>
<td>91%-95%</td>
<td>Less than 90%</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from the Global Initiative for Asthma guidelines, 2008.
worse—in a drowsy or confused patient, it can signal imminent respiratory arrest.\textsuperscript{1,2,4,6,9}

**Management strategies**

Because of great variability in clinical manifestations, severity, and response to treatments, your patient may need to be admitted to the ICU for close monitoring and aggressive treatment.\textsuperscript{1,2} Here are the recommended interventions for an asthma exacerbation:

- **Administer supplemental oxygen as soon as possible to achieve oxygen saturation of 90\% or greater.** A patient in an acute exacerbation inevitably suffers from hypoxia. Supplemental oxygen reduces V/Q mismatching, promotes bronchodilation, and reduces pulmonary vasoconstriction.\textsuperscript{6,10}

  If your patient also has chronic obstructive pulmonary disease (COPD), administering a large amount of supplemental oxygen may blunt his respiratory drive.\textsuperscript{4,8,11} These patients also are at risk of oxygen toxicity if they receive a fraction of inspired oxygen concentration ($F_{iO_2}$) greater than 0.5 to 0.6 for a prolonged period.\textsuperscript{11-14}

  However, the benefit of administering a large amount of oxygen (relieving hypoxemia in an acute asthma exacerbation) may outweigh the risks. Monitor your patient closely and titrate the oxygen to minimize the risks.

- **Administer rapid-acting inhaled bronchodilators as prescribed.** Beta$_2$-agonists stimulate beta$_2$ receptors on airway smooth muscles, relieving bronchoconstriction, reducing the work of breathing, and decreasing resistance to airflow. Short-acting beta$_2$-agonists (SABAs), quick-relief medications, are used to relieve bronchoconstriction, and are indicated for acute episodes and exacerbations.\textsuperscript{1} The commonly prescribed SABAs are albuterol [also known as salbutamol], terbutaline, levalbuterol, and pirbuterol.\textsuperscript{1,4,5,7,15}

  Nebulized salbutamol can be given intermittently or continuously. However, salbutamol that is given via inhaled or aerosolized routes may be unable to reach lower airways and lung tissues due to severe airflow obstruction and decreased tidal volume. If the patient’s initial response to SABA therapy is poor, consider I.V. beta$_2$-agonists.\textsuperscript{1}

  In the ICU, continuous I.V. infusion of terbutaline (the I.V. beta-agonist of choice in the United States) or salbutamol may be used to treat refractory asthma exacerbations.\textsuperscript{4,5,10,15}

  Subcutaneous injections of epinephrine or terbutaline may be considered if the patient fails to improve with nebulized beta$_2$-agonists.\textsuperscript{3-5,15} Nebulized or I.V. epinephrine also may be effective in treating a severe attack.\textsuperscript{7}

  Anticholinergics cause mild bronchodilation by inhibiting muscarinic cholinergic receptors and lowering intrinsic vagal tone of the airway. Administering inhaled ipratropium bromide in conjunction with a nebulized beta$_2$-agonist produces profound bronchodilation and significantly improves a patient’s pulmonary function, with minimal cardiovascular adverse reactions.\textsuperscript{1,3,5,6,10,16}

- **Administer systemic corticosteroids as prescribed.** Corticosteroids play a vital role in the suppression of airway inflammation. By inhibiting inflammatory cell migration and activation, and blocking late-phase reaction to allergens, corticosteroids reduce airway hypersensitivity, secretions, and edema. Systemic corticosteroids are an integral part of the first-line medication for moderate-to-severe asthma attacks.\textsuperscript{1,3} Prednisone, methylprednisolone, prednisolone, hydrocortisone, and dexamethasone are commonly prescribed and should be administered for 3 to 10 days. The dose reduction and duration of corticosteroid therapy are determined by the severity and chronicity of asthma.\textsuperscript{1,3-5,10,15,17}

  To prevent a relapse, the patient should be discharged with a 5- to 10-day course of oral systemic corticosteroids.\textsuperscript{1}

  Although corticosteroids are safe for short-term use, they can cause hyperglycemia, hypertension, fluid retention, hypokalemia, Cushing’s syndrome, acute psychosis, gastric ulcer, allergic reactions, and immune function suppression.\textsuperscript{1,4,10,15} Closely monitor patients on long-term corticosteroid therapy for adverse drug reactions.

- **Administer noninvasive positive-pressure ventilation (NPPV) as indicated.** NPPV enhances alveolar ventilation, improves gas exchange, and reduces the need for endotracheal intubation and mechanical ventilation. Applying extrinsic positive end-expiratory pressure (PEEP$_e$) with NPPV overcomes the effect of PEEPi, reduces the work of breathing, and rests fatigued respiratory muscles.\textsuperscript{5-8,18,19}

  No universal guidelines exist for when to implement noninvasive or invasive ventilation for asthma attacks, or for which ventilation strategy to use. However, NPPV may be used when aggressive treatments with bronchodila-
tors and corticosteroids don’t reverse a patient’s condition, and if the patient can tolerate the therapy, manage his own airway secretions, and doesn’t need immediate intubation.\(^6,7,17-19\)

NPPV can be administered via facial or nasal masks, using either continuous positive airway pressure or bilevel positive airway pressure. Be aware that inappropriately set PEEPs may worsen a patient’s hyperinflation. Other complications associated with NPPV include gastric distension, vomiting, aspiration, eye irritation, and stress ulcer.\(^7,18,20\) Some patients with severe asthma attacks can’t tolerate NPPV.\(^5,7\)

More and larger studies may be needed to determine the role of NPPV in managing severe asthma exacerbations.\(^9,21\)

- **Assist with endotracheal intubation and mechanical ventilation if indicated.** Your patient may need to be endotracheally intubated if he develops respiratory or cardiac arrest, severe hypoxia, progressive hypercapnia with profound acidosis, life-threatening dysrhythmias, hemodynamic instability, copious amount of sputum, exhaustion, or decreased level of consciousness.\(^1,4,5,8,10,22\)

Ventilating a patient with a severe asthma exacerbation often is challenging. Although mechanical ventilation improves gas exchange and rests fatigued respiratory muscles, it may lead to ventilator-induced lung injuries (VILI) such as barotrauma and volutrauma, and other complications. The ventilation strategy should aim to improve oxygenation, reduce dynamic hyperinflation, and PEEPi. The primary settings are for a low respiratory rate (8 to 10 breaths/minute), prolonged expiratory time (inspiratory-expiratory ratio of 1:2 to 1:6), and low tidal volume (4 to 8 mL/kg).\(^6,9,21\) Either volume control or pressure control ventilation can be used, depending on the healthcare provider’s prefer-
ence and experience. The mode and settings should be adjusted in response to significant changes in a patient’s condition.5-7,10,22

Low tidal volume or controlled hypoventilation elevates partial pressure of arterial carbon dioxide (PaCO₂). In patients with a severe asthma exacerbation, hypercapnia and respiratory acidosis are allowed to occur gradually in an effort to keep plateau pressure below 30 cm H₂O and reduce VILI.6,10,23,24

However, excessive hypercapnia causes intracellular acidosis and related cytotoxicity and induces pulmonary vasoconstriction that exacerbates preexisting pulmonary hypertension. Hypercapnia also causes cerebral vasodilation that increases intracranial pressure and leads to cerebral edema.24-26 No consensus exists on the upper limit of hypercapnia or the safe level of acidosis. The prevailing views are to keep PaCO₂ below 90 mm Hg and to maintain pH greater than 7.1.10,15,22,24,26

The use of PEEPi is controversial, because it reduces venous return and cardiac output, induces hypotension, and intensifies hyperinflation. Some healthcare providers advocate no PEEPi; others recommend applying a low PEEPi to reduce the work of breathing, improve patient-ventilator synchrony, and promote recovery of fatigued respiratory muscles.6,8-10,24,27

The current prevailing recommendation is to apply PEEPi at a level of about 80% of a patient’s PEEPi for spontaneously breathing patients.8,22,28 (The various methods used to detect and measure a patient’s PEEPi are beyond the scope of this article.)

• Assist with sedation and neuromuscular blockade if indicated. Patient-ventilator dyssynchrony is common in patients with a severe asthma exacerbation, because of extreme anxiety, agitation, and PEEPi. Sedation and paralysis rest fatigued respiratory muscles, reduce oxygen consumption and CO₂ production, ameliorate patient-ventilator dyssynchrony, improve patient comfort, and enhance the effectiveness of mechanical ventilation.6,29

Ketamine, midazolam, and propofol are commonly used for intubation and mechanical ventilation. Rocuronium or suxamethonium (neuromuscular blocking agents) often are prescribed to induce paralysis.7,10 Sedatives may lead to respiratory depression and delay weaning from mechanical ventilation. Neuromuscular blocking agents may cause myopathy or muscle weakness, so they should be used sparingly and only for a short time.5-9,24 Because corticosteroids may potentiate myopathy, they shouldn’t be administered concomitantly with neuromuscular blockers.30

• Administer adjunctive medications as prescribed. Magnesium sulfate causes smooth muscle relaxation. The Global Initiative for Asthma (GINA) guidelines recommend administering I.V. magnesium sulfate in certain cases, including adults who fail to respond to initial treatment.1 In combination with beta-agonists and systemic corticosteroids, this drug may be beneficial to patients in a severe asthma exacerbation.4,6,31 However, clinical evidence is inadequate to prove that magnesium improves a patient’s condition, and its therapeutic effect remains controversial.4,6,9,10,15,31,32

Methylxanthines such as aminophylline and theophylline were part of the standard treatment for severe asthma exacerbations in the past. But they can cause severe adverse reactions such as cardiac dysrhythmias, headache, nausea, tremor, seizures, and encephalopathy. Also, these drugs have a very narrow therapeutic range. As a result, aminophylline and theophylline are seldom prescribed, but may be used as second-line therapy for refractory asthma.2,4,10,17 Short-acting theophylline may benefit respiratory drive, although it doesn’t appear to provide additional bronchodilation over other SABAs.3

The GINA guidelines also recommend administering nebulized heliox as an adjunct treatment for severe exacerbations unresponsive to intensive treatments with SABAs, anticholinergics, and systemic corticosteroids.3 Using adjunct treatments may avoid the need for intubation. However, administering aminophylline or theophylline in the ED isn’t recommended.3

Other second-line options include inhaled anesthetic agents, mucolytics, and nitric oxide. Large studies are needed to evaluate the efficacy and safety of these agents and to determine their roles in treating severe acute asthma exacerbations.4,5,7,17

Your role
Closely monitor and regularly assess your patient to determine when therapy needs to be adjusted to improve patient response and minimize adverse reactions.1,2
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Assess and support the patient’s airway, breathing, and circulation and monitor his clinical status and vital signs. Help him assume a comfortable position (high Fowler’s or sitting, unless contraindicated by his hemodynamic status) and encourage him to relax. Maintain a calm environment and reassure the patient that you’re there to ensure his safety. Explain to him that agitation will worsen his breathlessness, and that relaxing will help reduce oxygen consumption and improve his condition.

Monitor your patient’s fluid balance. He may be dehydrated because of decreased oral intake and increased insensible fluid loss caused by agitation, increased work of breathing, and tachypnea. Administer fluid replacement as needed. Mechanically ventilated patients are at high risk of developing hypotension, so maintaining adequate intravascular volume is essential. However, be careful not to overhydrate your patient because of the risk of fluid volume overload and pulmonary edema.

If your patient needs NPPV, apply the facial or nasal mask properly and explain the therapy to your patient. Provide reassurance and emotional support to help him tolerate the therapy. Assess him regularly for the need for endotracheal intubation and mechanical ventilation. If he needs mechanical ventilation, monitor him for complications and take steps to identify and minimize VILI.

Teach the patient and his family about asthma control and prevention. (See Keeping asthma under control, every day) Review potential triggers and how the patient can control risk factors for acute attacks. Help him understand the importance of taking his medications and taking an active part in managing his disease.

Assess his ability to use his inhalers and to manage his asthma at home. Teach him to use a peak flowmeter and how to recognize trends that indicate an increasing risk of an acute attack. He should know the signs and symptoms of asthma exacerbations and when to seek medical attention. Arrange a follow-up appointment and refer him to an asthma self-management education program or asthma specialist as appropriate.

By understanding the management of an asthma exacerbation and how to empower patients to prevent future attacks, you can help your patient breathe easier.

REFERENCES


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Understanding ventilator waveforms: Erratum

In the article that started on page 43 in the January issue, waveform B was incorrectly positioned in Figure 5 on page 45. The corrected waveform is shown below. This error has been corrected in the online version of the article, which is available at www.nursingcenter.com.


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