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2nd Edition

Case-based Review in Critical Care Medicine

A Comprehensive Preparatory Book for the Examinee



Editors
Atul Prabhakar Kulkarni
Kapil Gangadhar Zirpe
Subhal Bhalchandra Dixit
Rahul Anil Pandit

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CHAPTER 5

Acute Severe Asthma

Babu K Abraham, Meghena Mathew

CASE STUDY

A 40-year-old gentleman with an ideal body weight of 70 kg comes to the emergency department with wheezing and breathlessness of 3 days' duration, which has worsened over the past 1 day. He is a known asthmatic on treatment with inhaled fluticasone and salmeterol (corticosteroid and long-acting β-2 agonist). On arrival, he is diaphoretic, tachypneic, tachycardic, and unable to speak in sentences. His saturation is 81% on room air. His vital parameters are heart rate of 140 beats/min, respiratory rate of 35 breaths/min, and blood pressure of 100/70 mm Hg. On auscultation, he has bilateral rhonchi.

Q1. What are the signs of acute severe asthma? What is the differential diagnosis that needs to be considered in this scenario?

Signs of acute severe asthma are a respiratory rate >30 breaths/min, use of accessory muscles of respiration, a pulse rate >120 beats/min, an oxygen saturation (SpO_2) on air that is <90%, inability to talk in sentences, a hunched posture, agitated appearance, and a peak expiratory flow rate (PEFR) ≤50% of predicted or best.[1] Some patients, with chronic asthma, may not be able to get their PEFR ever to that predicted for their height and weight. In such patients, we accept their best PEFR as normal for them and a reduction of 5% from their best PEFR is considered a sign of acute severe exacerbation.

The differential diagnosis for this clinical scenario should include pulmonary edema, viral pneumonia, chronic obstructive pulmonary disease (COPD) exacerbation, anaphylaxis, foreign body inhalation, and airway neoplasm compromising the airway.

Q1a. How is acute severe asthma different from uncontrolled asthma, difficult-to-treat asthma, and severe asthma?

Acute severe asthma is a different entity as compared to the other mentioned conditions. Both uncontrolled asthma and difficult-to-treat asthma will not have respiratory failure in their definition and will be more of an outpatient practice presentation.

Uncontrolled asthma[1] is a reason for persistent symptoms and exacerbations. It is defined by the presence of one of the following features—poor symptom control (defined as frequent symptoms or need for frequent reliever use, limitation of activity by asthma symptoms, and night waking due to asthma symptoms), frequent exacerbations (≥2/year) requiring oral steroids, or serious exacerbations (≥1/year) requiring hospitalization.

Difficult-to-treat asthma[1] is one that is uncontrolled despite the usage of medium or high-dose inhalational corticosteroid (ICS) with a second controller drug (e.g., long-acting β-2 agonist) or that requires oral steroid or that requires high-dose treatment to maintain good symptom control to reduce the risk of exacerbations.

Severe asthma[1] is a type of difficult-to-treat asthma where symptoms are uncontrolled despite adherence with high-dose inhalational steroids and long-acting β-2 agonist treatment and management of contributory factors or that can potentially worsen if the high-dose treatment is reduced.

Q1b. Does identifying asthma phenotypes help in improved management of patients with difficult-to-treat category?

Asthma phenotypes are patients with recognizable clusters of similar demographic, clinical, and/or pathophysiological characteristics.

Some of the most common asthma phenotypes described include allergic asthma, nonallergic asthma, adult-onset asthma seen in women predominantly, asthma with incomplete reversibility to bronchodilators, and asthma with obesity.

The other phenotypic classification of asthma is inflammatory phenotyping—type 2 or non-type 2. Type 2 is characterized by increased eosinophils and non-type 2 is characterized by elevated neutrophils. Eosinophilic inflammation is steroid responsive. Hence, type 2 patients

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with acute exacerbation will benefit with ICSs started at the earliest. However, during severe exacerbation, they might be refractory to even high-dose ICSs and they might benefit with oral corticosteroids.

Q1c. Describe the severity classification of asthma exacerbation.

Asthma severity is classified into mild, moderate, or severe.

The features of mild to moderate exacerbations include the ability to talk in phrases, absence of agitation, tachypnea, preference for a sitting position, pulse rate of 100–120 beats/min, absence of use of accessory muscles of respiration, SpO_2 on room air of 93–95%, and PEF > 50% predicted or the best value.

Severe exacerbation refers to when a patient is able to talk only in words, sits hunched forward, is agitated, respiratory rate of >30 breaths/min, pulse rate of >120 beats/min, accessory muscles are being used, SpO_2 on air is <90%, and PEF ≤ 50% predicted or best.

Q1d. Discuss the immediate management of this patient.

The immediate management of this patient should begin with an assessment of A (airway), B (breathing), and C (circulation). Look for altered mentation and a silent chest. Place a transcutaneous pulse oximetry and start oxygen via nasal cannulae or face mask to maintain SpO_2 around 93–95%. It may be difficult to do a PEFR monitoring in this situation and hence an arterial blood gas (ABG) analysis can be done. During an asthma exacerbation, partial pressure of arterial carbon dioxide ($PaCO_2$) is often <40 mm Hg. A $PaCO_2$ >40 mm Hg should be taken as an indication for respiratory fatigue.

The mainstay of acute severe asthma treatment is bronchodilator and steroid therapy. Start a short-acting β-2 agonist (SABA), a short-acting antimuscarinic agent (SAMA), and oral or intravenous corticosteroid. Salbutamol (2.5–5 mg) by nebulization every 20 minutes for three doses is a good choice for a SABA.[2] Evidence for pressurized metered-dose inhaler with a spacer during a severe and near-fatal asthma is less robust. After the initial repeated doses of nebulization, salbutamol (2.5–5 mg) should be continued every 1–4 hours, based on the clinical response to the initial therapy.[1-3] Effectiveness of salbutamol therapy is inversely proportional to the severity of bronchospasm.[4] There is a lot of evidence evolving now on the use of combination inhalational steroids—formoterol as an alternative to high-dose salbutamol demonstrating similar efficacy and safety profile. However, currently, challenges would be in terms of the availability of this combination as a nebulized solution and probably more data would be required to demonstrate better efficacy. Systemic steroids should be started early, especially when the response to initial bronchodilator therapy is poor. Both oral and intravenous steroids are equally effective. The doses recommended are either 1 mg/kg of prednisolone, 100 mg every 6th hourly[1,2] of hydrocortisone, or 0.5–1 g/kg of methylprednisolone in once-daily or divided doses. Their average duration of onset of action is 6–12 hours. Hence, the earlier the steroid is administered, the better the outcome[5] and less the relapse.[6] Intravenous route should be reserved for patients with impending respiratory arrest or for those who are already in respiratory arrest. High-dose ICSs should be given within the first hour after the presentation in patients not receiving systemic steroids.[1] In case of poor response to initial therapy, or persistent hypoxemia, or in those with forced expiratory volume in 1 second (FEV1) <25–30% done at the time of admission, intravenous magnesium is the next drug to be considered. Magnesium sulfate 2 g given intravenously over 20 minutes is the recommended dose.[1,2] It should be used with caution in patients with hypermagnesemia and renal failure.

Antibiotics are not routinely recommended unless there is a strong suspicion or evidence of lung infection as the trigger for asthma exacerbation.[1]

Frequent reassessment of the patient's response to therapy is absolutely essential. Failure to respond to medical therapy and respiratory fatigue warrants intensive care observation and early intubation and ventilation.[7] The decision to intubate and ventilate is purely based on clinical signs and must be made by an experienced physician without much delay.

This patient on admission has all features of acute severe asthma. The ideal strategy in the immediate management of acute severe asthma exacerbation is early recognition and timely intervention before the attack becomes life-threatening. The goal of management of asthma exacerbation is reversal of airflow limitation.

Arterial blood gas (room air) done shows pH—7.48, PCO_2—26 mm Hg, PO_2—50 mm Hg, HCO_3—28 mEq/L, and lactate—1.24.

Q2. Is there a role for noninvasive ventilation (NIV) or oxygen by high-flow nasal cannula (HFNC) in the management of acute severe asthma management?

The role of NIV in acute severe asthma, unlike in COPD, is not well proven.[8,9] If attempted, patients have to be carefully selected and closely monitored. NIV can probably be tried in patients who are not in impending respiratory failure, are hemodynamically stable, are neurologically intact, have no secretions, and are able to cooperate. A number of studies show potential benefit[8,10] in carefully selected groups, but larger clinical trials will be required before NIV can be recommended for all asthma patients.

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Similarly, the evidence for the use of HFNC in this setting is still debatable. The available data at this point of time is more in patients who present to the emergency room with acute dyspnea and hypoxemia of variable etiology. Compared to NIV, HFNC has shown to improve the sensation of dyspnea and comfort in this group of patients[11] with no difference in the intubation rate. At this point of time, no recommendation can be made on the use of HFNC in acute severe asthma in adults. There is emerging evidence for the safety and efficacy of HFNC in acute severe asthma in the pediatric population.[12]

Q2a. What is asthma–COPD overlap syndrome?

Patients with features of both asthma and COPD are characterized under this umbrella. Typically seen in the elderly population. They can have frequent exacerbations. Lung function studies will show persistent expiratory airway limitation. These patients will benefit from ICSs along with long-acting β agonist and long-acting muscarinic agonist as their maintenance therapy.[1] The management of this phenotype, when presenting with acute exacerbation, will be very similar to as mentioned earlier.

Despite having been initiated on intravenous steroids, nebulized SABA, nebulized SAMA, and intravenous magnesium patient shows no signs of symptomatic improvement. An ABG taken 1 hour into therapy shows pH—7.35, PCO_2—40 mm Hg, PO_2—64 mm Hg, and lactate of 3.5.

Q3. What are the indications for intensive care unit (ICU) admission in a patient with acute severe asthma? When would intubation and mechanical ventilation be considered? What are the rescue therapies that have been tried to prevent intubation and ventilation?

Indications for ICU admission in acute severe asthma exacerbations are lack of response to initial medical management, deteriorating PEF despite therapy, worsening oxygenation (<92%), worsening respiratory distress, respiratory fatigue with rising PCO_2, and anticipated need for mechanical ventilation.

The indications for intubation and ventilation are impending respiratory arrest from fatigue with altered sensorium that interferes with appropriate therapy, cardiac arrest, and respiratory arrest.

This patient warrants intubation as he is showing early signs of physical exhaustion with a rising trend of PCO_2 (from PCO_2 of 26 in the first ABG to PCO_2 of 40 in the second ABG) and lactic acidosis. Rising CO_2 is a sign of alveolar hypoventilation and in acute severe asthma, a normal PCO_2 is indicative of hypoventilation. The pathogenesis of lactic acidosis in acute severe asthma is unclear and multiple mechanisms have been proposed, including increased lactic acid production from anaerobic metabolism of the respiratory muscles that are under stress of increased work and decreased lactate clearance by the liver due to hypoperfusion/hypoxia and use of β-2 agonist.[4]

Many rescue therapies have been tried to avert intubation and break the bronchospasm in patients with acute severe asthma and they include the following:

Heliox

Heliox is a mixture of helium (He) and oxygen (O_2) mixed in a ratio of He:O_2—70:30 for medical use. Its density is less than that of air and it improves ventilation by reducing frictional resistance in the gas flow, encouraging laminar flow, and thus decreasing the work of breathing.[13] The low fraction of inspired oxygen (FiO_2) in the mixture limits its application in a hypoxic patient. In studies involving both intubated and nonintubated patients with both adult and pediatric population, the use of Heliox showed no significant difference in the recovery of bronchospasm.[14,15]

Intravenous ketamine

Ketamine has been tried in the treatment of refractory acute severe asthma both in children and adults[16] with mixed results. It is administered as a bolus dose of 0.5–1 mg/kg followed by infusion of 2 mg/kg/h under intensive care monitoring. Multiple case reports are available on the use of ketamine in severe asthma.[16,17]

Adrenaline

In addition to the β-2 agonist property, adrenaline through its action on other catecholamine receptors provides additional favorable effects in acute severe asthma. It reduces bronchial mucosal edema by its potent microvascular vasoconstrictive action. It causes further bronchodilation by decreasing the parasympathetic tone. It has also been shown to improve the partial pressure of arterial oxygen (PaO_2). Adrenaline given subcutaneously is the preferable route in asthma and can be tried in patients who do not tolerate or respond to nebulized medications. Use this drug with caution in patients >40 years of age, with known cardiac disease, and in pregnant women.

Intravenous leukotriene inhibitors

A randomized study comparing montelukast with placebo showed significant improvement in FEV1 in the montelukast group.[18] However, this study has been criticized for its methodology. The role of leukotriene antagonist is probably more applicable in asthma exacerbation secondary to aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs). The recommended dose is montelukast as a single 7–14 mg infusion over 5 minutes.

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Inhalational anesthetic agents

Halothane, isoflurane, and sevoflurane exert bronchodilatory properties by direct action on bronchial smooth muscle via calcium-dependent channels and by attenuating cholinergic tone. They reduce peak airway pressure within minutes of application and decrease pulmonary artery pressure. The dose of inhalational agents administrated needs to be titrated to clinical response. There are multiple case reports detailing the effects of isoflurane and sevoflurane in acute severe asthma.[19-21] But there are no randomized controlled trials (RCTs) to confirm their efficacy. Significant hypotension and myocardial depression are the most common side effects. Their use in the ICU is limited by cost, need for specialized equipment, need for intensive monitoring, and need for a scavenging system.

The use of intravenous β-2 agonist as a rescue therapy is no longer recommended.[1,2] Other therapies that have been reported in the literature are intravenous glucagon, nebulized clonidine, nebulized calcium channel blockers, and nebulized lidocaine.

Clinically, the patient has not responded to medical therapy and has progressed into a state of impending respiratory failure. A decision has been taken to intubate and ventilate him.

Q4. Describe the preparation required for intubating this patient. Mention the choice of sedation and premedications that are used.

Pathophysiology of asthma exacerbation includes increasing bronchospasm, increasing air trapping with worsening dynamic hyperinflation (DHI), depletion of catecholamines, and dehydration due to insensible losses. This leads to a patient who is exhausted with little physiological reserve. Post intubation there can be devastating consequences resulting from rapid fall in blood pressure and SpO_2. Anticipation and preparation are the keys to preventing or dealing with this deterioration.

Intubation should be planned and achieved as early as possible. Anticipate rapid desaturation in patients whose SpO_2 is ≤93%. Preoxygenate using a bag-valve-mask ventilation system or NIV. It may be difficult to achieve adequate preoxygenation due to their high residual volume and functional residual capacity.[22] When bagging, make sure that small tidal volumes, high-flow rates, and prolonged expiratory phase are used in an attempt to prevent breath stacking and generation of auto-positive end-expiratory pressure (PEEP). Place a couple of wide-bore intravenous cannulae and load them with intravenous fluid in anticipation of postintubation hypotension. Have inopressors kept ready for use if necessary. Have large-size endotracheal tubes (ETTs) (size 8 and above) and all the drugs required for modified rapid sequence intubation (RSI) ready.

Nasotracheal intubation is to be avoided as nasal polyps are common in asthmatics. Orotracheal intubation with larger diameter ETT helps with the decrease in airway resistance, toileting of airways, and therapeutic bronchoscopy if needed.

The preferred approach of intubation would be modified RSI. Pretreatment with lidocaine may be indicated in patients who have not received any bronchodilators. Opioid and barbiturates help to blunt sympathetic response secondary to intubation; however, there is a small risk of histamine release. Ketamine and propofol are considered the drugs of choice for induction.[23] Ketamine is the preferred drug as it does not cause hypotension (dose: 1–2 mg/kg intravenous bolus followed by an infusion at 0.5–0.75 mg/kg/h). It may be contraindicated in patients with hypertension, ischemic heart disease, raised intracranial pressure (ICP), and preeclampsia. Propofol too has been shown to have bronchodilator effect. However, its greatest drawback is hypotension. Other drugs such as midazolam and etomidate have been used as induction agents.

There is no difference between using succinylcholine or a nondepolarizing agent for paralysis.[7]

The patient has been successfully intubated and is being hand ventilated. Arrangements are being made to connect him to mechanical ventilation ***(Flowchart 1)****.*

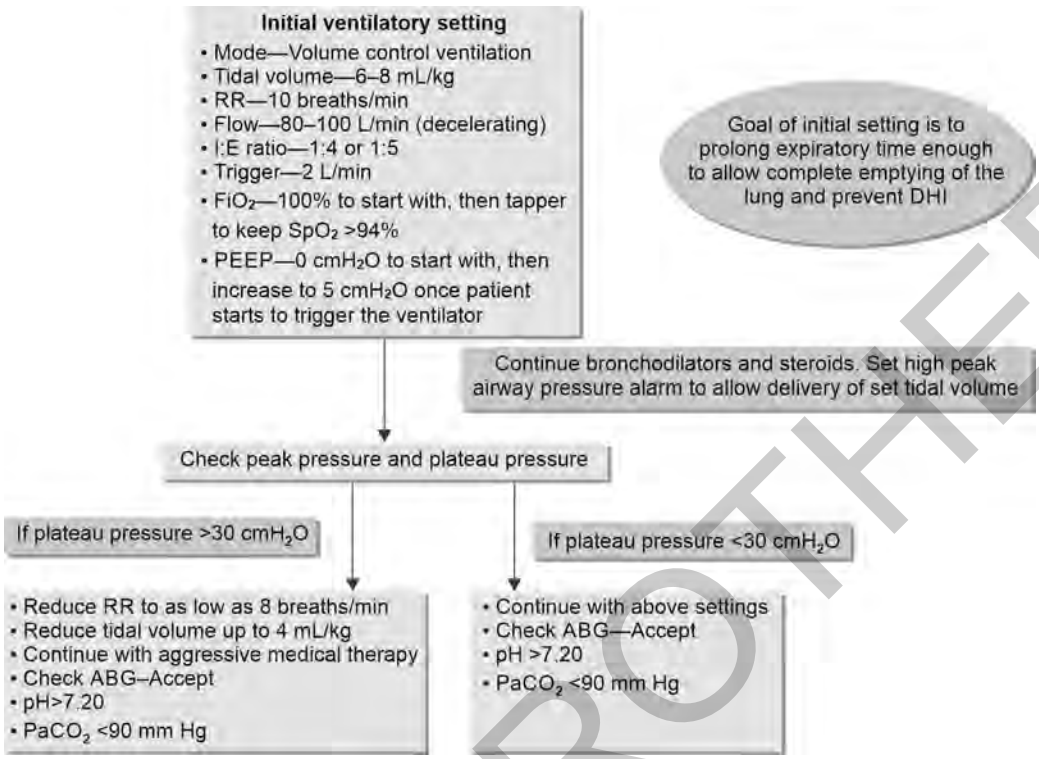
Q5. Discuss the management of sedation and paralytic agents post intubation and describe the initial ventilator settings in this patient.

Once intubated, sedation is essential to improve patient comfort, decrease tissue oxygen consumption, control respiratory rate, decrease air trapping, and provide better patient–ventilator synchrony. To provide adequate analgesia with amnesia and adequate suppression of respiratory rate, a combination of an opioid (fentanyl: 1–2 μg/kg/h) with a benzodiazepine (midazolam: 2–10 mg/h) can be used. The use of neuromuscular blocker should be reserved for patients who cannot be adequately ventilated with deep sedation alone and if used, it should be restricted to <24 h. The drug of choice would be cisatracurium in a dose of 1–2 μg/kg/min due to its better cardiorespiratory effects and nonhepatic or renal elimination.[7]

The objectives of setting a ventilator in a patient with acute severe asthma are to prevent breath stacking and reduce DHI. This is achieved by keeping a low tidal volume, a low respiratory rate, higher inspiratory flows, and a short inspiratory time (Ti). The occurrence of hypotension immediately after initiation of mechanical ventilation can be due to a combination of DHI, hypovolemia, vasodilatory effect of induction drugs, and tension pneumothorax.

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Flowchart 1: Initial ventilator setting after intubating asthmatic patient.



(ABG: arterial blood gas; DHI: dynamic hyperinflation; FiO_2: fraction of inspired oxygen; $PaCO_2$: partial pressure of arterial carbon dioxide; PEEP: positive end-expiratory pressure; RR: respiratory rate)

First, choose the mode in which the patient should be ventilated. There is no single preferred mode of ventilation for these patients. Outcomes have been similar with both volume preset and pressure preset modes.[24] However, volume preset mode is preferred as it has been most studied. The suggested initial ventilator settings in a volume-controlled ventilation are a tidal volume of 6–8 mL/kg of ideal body weight, respiratory rate of 10 breaths/min, peak inspiratory flow of 60 L/min with constant flow pattern or 80–90 L/min with decelerating flow pattern, an inspiratory to expiratory ratio (I:E ratio) of 1:4 to 1:5, FiO_2 of 100%, and a PEEP of 0 cm Hg, as long as the patient is sedated and paralyzed.

The FiO_2 has to be gradually reduced from 100% with the goal to maintain saturation >94%. As the sedation is lightened and the patient starts to trigger the ventilator, the trigger sensitivity should be kept at 2 L/min. Monitor the plateau pressure closely and keep it <30 cmH_2O. Set the peak pressure alarm above the patient's peak pressure seen on the ventilator to avoid hypoventilation. The low respiratory rate and tidal volume (6 mL/kg) used for preventing DHI in asthma ventilation can cause hypercapnia. It is acceptable to allow hypercapnia with a safe limit set at pH above 7.2 and PCO_2 at 90 mm Hg.[25,26] "Permissive hypercapnic ventilation" is the terminology used for this type of ventilator setting.

After initiating mechanical ventilation, always do a quick bedside examination looking for symmetrical chest rise, bilateral equal air entry, and evaluation of vital parameters looking for hypotension and hypoxia.

After a couple of hours of setting the ventilator, the high-pressure alarm starts to go off frequently.

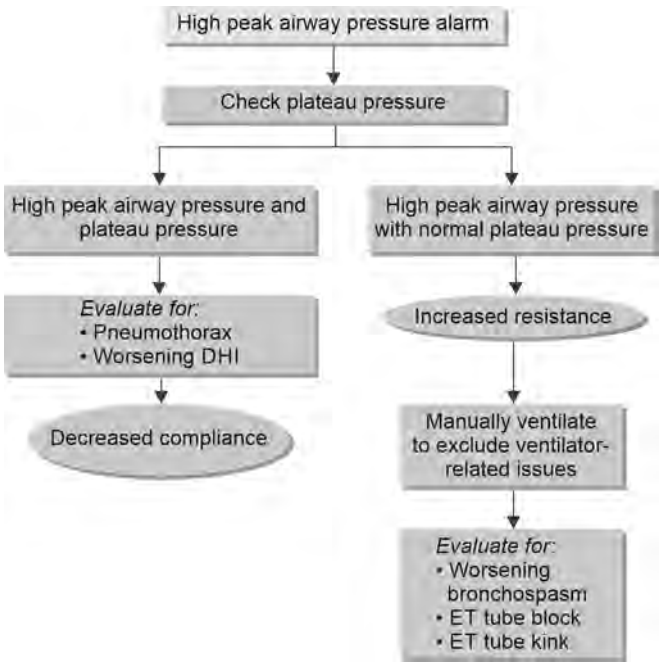
Q6. Explain how to troubleshoot high-pressure alarms in this patient.

A peak inspiratory pressure of >80–100 cmH_2O is not unusual in patients with severe asthma. Set the high-pressure alarm limit at 100 cmH_2O to facilitate delivery of the set tidal volume. The most common cause for high-pressure alarm would be worsening bronchospasm. However, other causes such as decreased lung/pleural compliance (e.g., hyperinflation, pneumonia, pneumothorax) and patient–ventilator dyssynchrony need to be looked for.

To troubleshoot high peak airway pressure alarm, it is essential to differentiate between an airway resistance problem and a lung compliance issue. Peak pressure is the sum of airway resistance pressure, PEEP, and lung elastic pressure, while plateau pressure, obtained by applying an inspiratory pause, reflects alveolar pressure. When both peak pressure and plateau pressure increase, it is indicative of a compliance problem. Evaluate the patient for a lung

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Flowchart 2: Evaluating high peak airway pressure alarm.



(DHI: dynamic hyperinflation; ET: endotracheal)

parenchymal, pleural, and chest wall or a diaphragmatic problem. An elevated peak airway pressure with a normal plateau indicates an airway resistance problem.

Then evaluate the patient for worsening bronchospasm, ETT block or kink **(Flowchart 2)**.

All of a sudden, he develops hypotension (80/50 mm Hg) with worsening hypoxia, which rapidly deteriorates into a pulseless electrical activity (PEA).

Q7. Enumerate the possible causes for this sudden setback and discuss how the cardiac arrest can be managed.

Advanced cardiac life support (ACLS) algorithm needs to be initiated for the PEA cardiac arrest while simultaneously looking for a reversible cause.

The cause for PEA cardiac arrest that is specific for patients with acute severe asthma on mechanical ventilation is DHI, which is accentuated by positive pressure ventilation. DHI happens in asthma due to incomplete exhalation leading to air trapping. This further worsens if the ventilator is not set properly giving sufficient expiratory time for the lung to empty.

Positive pressure ventilation by virtue of creating a change in heart–lung interaction can cause cardiac arrest by decreasing the preload to the right ventricle, increasing pericardial pressure, and increasing total pulmonary vascular resistance and right ventricular strain. The change in transpulmonary pressure can predispose to tension pneumothorax. The other factors that worsen hemodynamic instability are sedation-induced vasodilatation and preexisting hypovolemia.

If DHI is suspected, a trial of hypoventilation (delivering 2–3 breaths/min of 100% oxygen for a few minutes) can be done both as a diagnostic and therapeutic intervention. Tension pneumothorax could be a cause for PEA arrest and if clinically suspected, a needle thoracostomy followed by intercostal drainage needs to be done. Other causes of PEA cardiac arrest include electrolyte abnormalities (including lethal hyperkalemia if succinylcholine was used for intubation of a patient with respiratory acidosis), severe acidosis, myocardial ischemia (particularly if high-dose β-2 agonists were used systemically), and ETT displacement, kinking, or plugging should be recognized and treated appropriately.

The patient has been successfully resuscitated out of PEA with a return of spontaneous circulation (ROSC) of <2 minutes. However, he continues to require a FiO_2 of 1 to maintain an SpO_2 >88%.

Q8. What are the possible causes of postintubation hypoxia and how can they be managed?

The common causes for postintubation hypoxia are right main stem intubation, ETT displacement, ETT blockage, leakage of air around the ETT, pneumothorax, gastric distension decreasing respiratory system compliance, mechanical malfunction of the ventilator apparatus, aspiration, and worsening bronchospasm. Identification of these complications is essential as management differs and the initial assessment includes clinical examination, chest X-ray, and use of bedside ultrasonogram.

Right main stem intubation

Clinical examination usually clinches the diagnosis. Diminished chest movement of the left hemithorax and auscultation revealing asymmetrical breath sounds with absent air entry on the left is usually diagnostic. Postintubation chest X-ray will confirm the diagnosis.

Pneumothorax

There should be a high level of suspicion for a pneumothorax. Look for increased airway pressures and unilateral absent breath sound with hyperresonant percussion note. Bedside ultrasonography (USG) examination can be helpful. An emergent needle thoracostomy followed by intercostal drainage should be performed immediately as this could progress to tension pneumothorax if left unattended.

Endotracheal tube displacement

Assess the position of the tube at the lip level. A chest X-ray or direct visualization with a laryngoscope can be done to evaluate any displacement. Once identified, direct visualization and repositioning under sedation are advised.

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Endotracheal tube blockage

Decreased bilateral air entry, difficulty to deliver set tidal volume, and a rising peak pressure should alert one with the possibility of a tube block. This condition also warrants replacement with a new tube.

Endotracheal tube cuff leak

A cuff leak can be diagnosed by an audible air leak and inability of the ventilator to deliver the set tidal volume. If there is a cuff leak, replace the tube.

Gastric distension

Gastric distension can decrease respiratory compliance and usually occurs following the application of NIV or vigorous bag-valve-mask ventilation prior to intubation. It can be diagnosed by clinical examination and is treated by decompressing the stomach by placing a nasogastric tube.

Pneumonia

Suspect pneumonia if the patient has a fever and thick purulent sputum. A chest X-ray will usually help with the diagnosis. Antibiotics need to be initiated if pneumonia is suspected.

Finally, a quick check of the ventilator by placing a test lung will help in evaluating the functioning of the ventilator.

The patient's hypoxia improves with therapy but he continues to be deeply sedated and paralyzed to facilitate mechanical ventilation. On performing an end-inspiratory hold and end-expiratory hold, the plateau pressure is recorded to be 40 cmH_2O with a peak pressure of 80 cmH_2O, and intrinsic PEEP of 18 cmH_2O.

Q9. What do the above pressure measurements mean and could this patient have DHI?

This patient has both elevated plateau pressure and peak airway pressure, which is a sign of decreased lung compliance (*see* Q6). In a patient with pure asthma, decreased lung compliance could be an indicator of impending DHI.

Dynamic hyperinflation happens when inadequate expiratory time leads to incomplete emptying of alveoli with associated trapping of some amount of the delivered tidal volume in the alveoli at the end of expiration. With every breath, more and more air is entrapped in the alveoli, thus making the pressure within the alveoli to go up and the compliance of the lung to come down. This can be assessed at the bedside by measuring the plateau pressure which is a surrogate of alveolar pressure, intrinsic PEEP which is the measure of auto-PEEP, and end exhalation volume which tells us about the amount of gas trapped in the alveoli.

Measuring plateau pressure helps in assessing for DHI[27,28] and a pressure >30 cmH_2O is associated with increased lung complication.[29] Measuring the intrinsic PEEP by doing an expiratory pause maneuver has no definite cutoff value, which has been associated with increased risk for DHI since it can be falsely low in patients with severe airway obstruction due to airway closure.[30] A flow time scalar with an expiratory flow limb that fails to return to baseline before the initiation of subsequent breath is indicative of presence of an auto-PEEP. DHI can also be monitored by looking at the total exhaled volume of gas during a 20–40 seconds apnea (end-inspiratory lung volume, i.e., VEI). A VEI >20 mL/kg has been associated with barotrauma and adverse heart–lung interactions. Since this technique cannot be done easily at the bedside, it is not used routinely.[7] Many experts agree that complications are rare when plateau pressure is <30 cmH_2O and intrinsic/auto-PEEP is <15 cmH_2O.[31]

Q10. How can the ventilator settings be modified based on these findings (given above)?

Three ventilator strategies that can be employed to reduce DHI are (1) reduction of respiratory rate to 8–10 breaths/min, (2) reduction of tidal volume to 4 mL/kg, and (3) shortening of inspiratory time to allow greater time for exhalation.

Reduction of respiratory rate has the greatest impact on the reduction of DHI. However, setting a respiratory rate of <10 breaths/min would require deep sedation and may be even paralytic agents. The reduction of tidal volume is limited by the inverse relation it has with dead space. The impact of reducing minute ventilation below 10 L/min on DHI is very minimal.[32] The aim is to achieve sufficient expiratory time (I:E ratio of 1:4 to 1:5) to allow the lungs to empty. This can be monitored by observing the flow time scalar and allowing the expiratory flow wave to touch the baseline before the initiation of the next breath. If a decelerating flow pattern is being used, in volume control ventilation, changing to a square wave flow pattern would increase the flow rates further and prolong the expiratory time further.

These interventions need to be monitored closely. A plateau pressure decreasing below 30 cmH_2O and a flow time scalar showing expiratory flow touching baseline are signs of resolving hyperinflation. Despite optimizing these maneuvers, if DHI does not settle, it clearly indicates that there is severe bronchospasm and this can be overcome only with aggressive bronchodilator therapy and steroids.

Q11. What is the role of extrinsic PEEP in this patient?

Extrinsic PEEP has no role in patients with acute severe asthma who are sedated/paralyzed and have no spontaneous efforts. The application of external PEEP in such patients has to be done with caution as it can worsen lung compliance and exacerbate the DHI. A low level of PEEP (5 cmH_2O), in patients who are triggering the ventilator or are spontaneously breathing, can decrease the inspiratory work of breathing and help improve ventilator triggering by

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decreasing the pressure gradient needed to overcome auto-PEEP.[33] Every time extrinsic PEEP is added or increased, the plateau pressure needs to be checked to make sure there is no worsening of air trapping and lung compliance.

After optimizing the ventilator settings, an ABG is repeated and shows a pH—7.28, PCO_2—67 mm Hg, PO_2—75 mm Hg, and HCO_3—29 mEq/L.

Q12. What are the contraindications to permissive hypercapnia?

Contraindications to permissive hypercapnia are recent myocardial infarction, pregnancy, and raised ICP.

Despite the direct myocardial depressant effect, hypercapnia can cause an increase in heart rate by inducing sympathetic hyperactivity. This can predispose to arrhythmias, which are poorly tolerated in acute myocardial infarction. It can also cause pulmonary artery vasoconstriction and lead to hemodynamic instability in patients with right heart dysfunction.

Hypercapnia causes increased ICP by its direct cerebral arteriolar vasodilatory effect, which leads to increased cerebral blood flow.

In pregnancy, high CO_2 can cross the placental barrier and cause fetal acidosis, shifting the oxygen dissociation curve to the right, and decreasing the fetal hemoglobin affinity for oxygen.

Despite optimizing the mechanical ventilator settings and aggressive bronchodilator therapy, the patient continues to have severe bronchospasm and is difficult to ventilate.

Q13. Discuss the management of refractory bronchospasm.

Unfortunately, despite aggressive bronchodilator therapy, some patients continue to have refractory bronchospasm, hypoxia, and can be difficult to ventilate. In such patients, the following therapies can be tried.

Heliox

See Q3

Inhaled general anesthetic agents

See Q3

Ketamine infusion

See Q3

Bronchoscopic removal of impacted mucus

This technique has been anecdotally reported to decrease airway pressures and improve gas exchange.[34] It may be considered in patients with persistent high peak airway pressure and difficulty in weaning from a mechanical ventilator. The concern with this technique is worsening bronchospasm.

Extracorporeal life support

Extracorporeal life support (ECLS) has been attempted in the management of severe asthma. Venovenous ECLS has been used for extracorporeal CO_2 removal. Probably an ideal situation for its use may be patients with severe hyperinflation, profound respiratory acidosis, and hemodynamic instability who have not responded to maximum medical therapy. Evidence for routine ECLS use in acute severe asthma is still lacking.[35]

Methylxanthines

Drugs in this group were once the first line in the management of acute severe asthma. However, they have been shown to have no additional benefits when used along with β-2 agonist in an acute setting.[1-3] They are not very potent bronchodilators and have an increased incidence of adverse effects when used in combination with SABA.[36] However, patients who are on long-term oral theophylline can be continued on the same dose after checking baseline serum theophylline levels.

Intravenous β-2 agonist

Routine use of parenteral β-2 agonist is no longer a part of treatment for adult asthma exacerbation guidelines.[1] Use has been associated with increased adverse effects such as tachycardia, myocardial injury, and lactic acidosis.

After 24 hours of aggressive bronchodilator therapy and optimized mechanical ventilation, the patient's bronchospasm has settled. He is weaned off paralytic agents and sedation. He wakes up appropriately.

Q14. What criteria need to be met before he can be weaned off mechanical ventilation and extubated?

There are no guidelines for weaning and extubating patients specifically recovering from acute severe asthma. One safe approach is to perform a spontaneous breathing trial once the patient is awake, bronchospasm has settled, and $PaCO_2$ has normalized. Once airway resistance is <20 cmH_2O, auto-PEEP is <10 cmH_2O, and there is no evidence of neuromuscular weakness, extubation should be attempted.[7] Post extubation, observation for 12–24 hours in ICU would be prudent.

On attempting a trial of spontaneous breathing, he goes into respiratory distress and needs to be placed back on the control mode of ventilation.

Q15. Discuss the causes of failure to wean from mechanical ventilation.

Factors that cause weaning failure are persistent bronchospasm, impaired respiratory mechanics, cardiac dysfunction, neuromuscular weakness, electrolyte imbalance, metabolic and endocrine factors, and cognitive

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dysfunction. These need to be looked for and treated appropriately. Persistent bronchospasm is a very common cause for weaning failure and probably needs revaluation of medical therapy. Severe muscle weakness is seen in up to 15% of patients who undergo mechanical ventilation for severe asthma.[37] The pathogenesis of this myopathy is unclear and has been attributed to the combined use of steroids and neuromuscular paralytic agents.[37,38] Use of deep sedation alone can also lead to muscle inactivity that results in myopathy, which is generally reversible.

Q16. Is there a role for extracorporeal membrane oxygenation (ECMO) if he fails multiple attempts at weaning in asthma?

Evidence for the use of ECMO in acute severe asthma is limited.[39] It can be considered when the severe bronchospasm is refractory to maximal medical therapy and ventilation becomes difficult with significant hypercapnia and hemodynamic instability. ECMO can also be considered to prevent complications of mechanical ventilation such as DHI, pneumothorax, and in situations of ventilator-related complications such as severe air leaks and ventilator-induced lung injury. At this point of time, due to the lack of strong evidence, the need for intensive monitoring, the financial implications, and the complications associated with its use, ECMO can only be recommended as a rescue therapy for acute severe asthma. Multiple case reports are available showing good success rates with ECMO therapy in asthma.[40,41] A recent retrospective observational cohort study[42] evaluated patients with asthma exacerbations and respiratory failure, who were intubated after medical management. These patients were treated in an ECMO-capable hospital. This study compared patients who received ECMO with patients who did not get ECMO showed decreased mortality, at the expense of higher hospital costs and suggested that ECMO could be an important salvage therapy. However, well-defined clinical trials only can better answer this question.

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About the Editors

Atul Prabhakar Kulkarni MD (Anesthesiology) FISCCM PGDHHM FICCM is currently Professor and Head, Division of Critical Care Medicine, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. A recognized postgraduate teacher for MD (Anesthesiology), he is now a Teacher for DM (Critical Care Medicine). A postgraduate diploma holder in Postgraduate Diploma in Health and Healthcare Management, he has received training in Intensive Care at St George Hospital, Sydney, Australia in 2002–2003. He has participated in many conferences on critical care and anesthesiology at international, national, and state levels. He was the Organizing Secretary for Anesthesia Review Course since 2004 to 2018. He was the Treasurer of Indian Society of Critical Care Medicine during 2004–2010. He was later General Secretary of Indian Society of Critical Care Medicine (2010–2014) before he took over as the President of Indian Society of Critical Care Medicine (2015–2017) and the Chancellor, Indian College of Critical Care Medicine. He was a Member of the Editorial Board of Indian Journal of Critical Care Medicine (IJCCM) and Reviewer for IJCCM and Indian Journal of Anesthesia (IJA). He is also the Past President of Association of South Asian Association for Regional Cooperation (SAARC) Critical Care Societies and Secretary General. After serving as the Honorary Secretary General of the Asia Pacific Association of Critical Care Medicine, he took over as the President of the prestigious organization. Currently, he is the Editor-in-Chief of the IJCCM.

Kapil Gangadhar Zirpe MBBS MD (Chest) FICCM FCCM FSNCC is the Head, Department of Neuro-Trauma Intensive Care Unit, Grant Medical Foundation, Ruby Hall Clinic, Pune, Maharashtra, India. He has completed MBBS and MD (Chest Medicine) from Dr VMMC Medical College, Solapur, Maharashtra affiliated to Shivaji University, Kolhapur, Maharashtra. He was conferred with Fellowship of Indian College of Critical Care Medicine (FICCM) by Indian Society of Critical Care Medicine (ISCCM) in 2013 and Fellowship of American College of Critical Care Medicine (FCCM) in 2016 by Society of Critical Care Medicine (SCCM). He was conferred with Fellowship of College of Neurocritical Care Medicine (FSNCC) by Society of Neurocritical Care (SNCC), India in 2021. He is the President of Association of SAARC Critical Care Societies (ASARCC). He is the Past President of SNCC and the Past President of ISCCM, Mumbai. He was a Council Member of World Federation of Societies of Intensive and Critical Care Medicine (WFSICCM). He is Council Member of Asia Ocean Chapter of Neurocritical Care Society (NCS). He is an Editorial Board Member of Indian Journal of Critical Care Medicine (IJCCM) and Reviewer for IJCCM, Indian Journal of Medical Research (IJMR) and IJCC.

Subhal Bhalchandra Dixit MD (Medicine) FCCM FICCM FICP is the Director, Department of Critical Care ICU, Sanjeevan and MJM Hospital, Pune, Maharashtra, India. He has held major posts at the national level including Past President, ISCCM, and Past Chancellor, ICCCM. Currently, he is Treasurer, Sepsis Forum of India. He has a special interest in critical care and specializes in critical care nutrition, sepsis, and ventilation. He has authored numerous chapters in various textbooks and has many national and international publications and original published research. He is also the Chief Editor of various books in critical care and nutrition.

Rahul Anil Pandit MD FJFICM FCICM IFCCM EDIC DA is the Chair of Critical Care Medicine, Sir HN Reliance Foundation Hospital and all other Reliance Foundation Healthcare Initiatives, Mumbai, Maharashtra, India. He is Visiting Consultant to Bathurst and Wagga Wagga Base Hospital, NSW, Australia. He is the Vice President of Indian Society of Critical Care Medicine and the Chairman of the Indian Society of Critical Care Medicine (ISCCM), Mumbai Branch. He is a Teacher for the College of Intensive Care Medicine of Australia, ISCCM and National Board of Examinations. He has published many papers and written several chapters.

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