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Review Article

Perioperative Anaesthetic management in Asthma

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Abstract

Asthma is a chronic disorder of airway characterized by hypersensitiveness to a variety of triggers. With a potential to cause an anaesthetic disaster during perioperative period, acute episode of asthma can lead to life threatening complications. It is important for an anaesthesiologist to make a positive difference in the outcome of the asthmatic patient by stepwise approach pre, peri and post operatively.

This review takes the anaesthetist's perspective to discuss the approach to asthma, safe practices and special considerations to be followed in asthmatics. This article focuses on the pathophysiology, pharmacotherapy and perioperative management in acute bronchial asthma.

Keywords: Bronchial Asthma, Anaesthesia, Anaesthetic management, perioperative bronchospasm

1.Introduction

Asthma is a major public health issue with high and increasing prevalence rates [1] and a concomitant increase in morbidity and mortality.[2] Studies have shown that the lifetime prevalence of asthma among adults is 11%[3] and it is even higher among children.[4] These data contribute to make challenging adverse events due to asthma.

"Asthma is a chronic disorder of the airway in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyper responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread, but variable airflow obstruction within the lung that is often reversible either spontaneously or with treatment."[5]

Bronchospasm the clinical feature of exacerbated underlying airway hyper reactivity has the potential to become an anesthetic disaster. During the perioperative period, bronchospasm usually arises during induction of anesthesia but may also be detected at any stage of the anesthetic course. Accordingly, prompt recognition and appropriate treatment are crucial for an uneventful patient outcome. Perioperative bronchospasm (i.e., the clinical expression of exacerbated underlying airway reactivity) may be associated with type E immunoglobulin (IgE)-mediated anaphylaxis or may occur as an independent clinical entity, triggered by either mechanical and/or pharmacologic factors. Whatever the clinical circumstances, different triggers are identified in the occurrence of bronchospasm during induction.[6] Therefore, in order to avoid increasing the risk of perioperative complications, a good understanding of asthma pathophysiology, an adequate preoperative evaluation and optimization of the patient's condition, allied with the best pharmacological and technical approach are imperative.

In view of these, we attempted this review to outline available data and criteria for the anesthetic management in asthmatic patients.

2. Pathophysiology

Asthma implies a wide phenotype range of severity, chronicity, persistence and response to therapy. The severity of the disease process is related

to the severity of airway inflammation, which governs hyper responsiveness, the degree of obstruction and symptomatology.

Bronchoconstriction results from contraction of bronchial smooth muscle induced by a myriad possible stimuli, including intrinsic factors, allergens, exercise, stress, or cold air. Vagal and sympathetic factors directly modulate airway tone. Inflammatory oedema and mucous plugging exacerbate airflow limitation and progressively impair the response to bronchodilator therapy. Airway remodelling, thickening, and abnormal communications between the injured airway epithelium and the pulmonary mesenchyme confer resistance to corticosteroid therapy as well.[7] Airway smooth muscle changes have been implicated in chronic, poorly responsive bronchospastic disease—both as a mechanical and as an inflammatory mediator.[8]The immunologic-inflammatory pathways involved in the pathogenesis of asthma are complex and include lymphocytes (both Th1 and Th2), immunoglobulin E, eosinophils, neutrophils, mast cells, leukotrienes, and cytokines. These pathways are triggered and modified by extrinsic and environmental factors such as allergens, respiratory infections, smoke and occupation-related exposure.[9] Thus, asthma ultimately represents a dynamic interaction between host and environmental factors.

3. Pharmacotherapy

Historically, medications for asthma have been classified according to mechanism and target. More recently, a short- vs. long-term relief schema has become popular, particularly as newer drugs defy the old classification.[10]This is considered to be more amenable to patient compliance and simplifies the perioperative approach to therapy.

3.1 Quick-acting drugs

Quick-acting β2-selective adrenergic agonists provided by metered-dose inhalers (MDIs) are the mainstay for fast relief of bronchoconstriction. Examples of this class of drugs are albuterol (salbutamol- 1.25 to 5 mg in 3ml saline Q 4-8hr) and levalbuterol (levosalbutamol 0.63-1.25mg Q 4-8hr). Their onset of action occurs within 5 min, peak effect is within 1 hr, and their duration of action is 4–6 hr. Patients with poor inhalation technique should use a valved holding chamber (spacer). Recommended only for short-term relief of symptoms or before known triggers such as exercise. Side-effects such as tremor, anxiety, palpitations, and tachycardia occur but are not common at standard doses.[10] Parenteral administration of short-acting β2-agonists is discouraged because of slow onset time, diminished potency and considerably greater systemic adverse effects.

3.2 Long-acting drugs

Long-acting β2-selective agonists, such as arformoterol, formoterol, and salmeterol, (12 µg/blister every 12hr) provide bronchodilation for >12 h and are largely free of side-effects. The long-acting agents do not suppress inflammation and should not be used without anti-inflammatory treatment for the control of asthma.[12]

Inhaled corticosteroids, for example, beclomethasone (40 µg twice daily two inhalations), budesonide (0.25mg/2ml twice daily) and fluticasone (88µg twice daily), are the cornerstones of therapy to stabilize and improve persistent asthma. Their consistent use has probably contributed to the decreased morbidity and mortality observed in asthma, nonetheless, inhaled corticosteroids are suppressive rather than curative. No clinically important adrenal suppression has been found with their administration in low to moderate doses. No significant therapeutic differences appear to exist between different formulations.[12]

Parenteral steroids such as hydrocortisone (200mg IV stat) and methyl prednisolone (40-80mg IV per day) remain a mainstay of the treatment of acute asthma. However, their beneficial effect on airway mechanics can take 4–6 h in acute bronchospasm, a noteworthy point in the poorly controlled asthmatic requiring urgent or emergent surgery.[13] Adrenal suppression, infection, delayed healing, hyperglycemia and fluid retention are common complications of prolonged therapy. However, delayed wound healing and increased infection have not been observed in asthmatic patients treated with perioperative systemic steroids.[14] Patients who have been taking systemic corticosteroids for >2 weeks during the prior 6 months should be considered at risk for adrenal suppression in the setting of severe acute disease, trauma or major surgery.[12]

Leukotriene modifiers, such as montelukast (10mg hs), zafirlukast (40mg per day) and zileuton (2400mg per day), inhibit the leukotriene pathway, a mediator of bronchoconstriction. Evidence of their beneficial effect on inflammation is conflicting. They are not useful for acute treatment of bronchospasm. Leukotriene modifiers are second-line to steroids in the management of chronic asthma.[12]

Anticholinergic bronchodilators such as ipratropium (0.5mg Q 20minutes for three doses) have a more limited role in the therapy of acute asthmatic bronchospasm than β2-selective agonists. Indications for their use include, as implied, patients

with COPD, but also patients who are intolerant of β2-selective agonists, or who are severely asthmatic, or have β-blocker-induced bronchospasm.[15] Anticholinergic agents dry airway secretions. Although controversy exists as to whether this improves inflammation or worsens inspissation, it certainly decreases airway hyper responsiveness, an important consideration for the anaesthetist.[12]

4. Preoperative evaluation

A thorough history and physical examination provides the anesthesiologist with information that allows for appropriate identification of level of disease, degree of symptom control and anesthetic risk stratification. Review of baseline exercise tolerance, hospital visits secondary to asthma (including whether endotracheal intubation or IV infusions were required), allergies and previous surgical/anesthetic history is essential. The patient's medication regimen should be reviewed and provides important clues as to level of disease severity. Patients should be queried as to new medications, recent changes in the frequency or dose of medications and the level of disease control on their current medication regimens. This thorough review of an asthmatic's history and the appropriate preoperative preparations can significantly reduce the risk of adverse outcomes.[16]

The risk of intraoperative bronchospasm, one of the most feared complications of asthma, can be increased by the presence of atopy, eczema, allergic rhinitis, and other conditions of chronic inflammation.[17] A family history of asthma and atopy should be sought and is also a marker of increased perioperative risk.[18] Smoking or exposure to second-hand smoke contributes to poor asthma control and is also an independent risk factor for adverse respiratory events under general anesthesia.[19]If time permits, the patient should be advised to stop smoking for 2 months prior to elective surgery.[20]

5. Physical examination

Physical examination should include vital signs and assessment of breath sounds, use of accessory muscles, and level of hydration. The presence of labored breathing, use of accessory muscles and prolonged expiration time suggest poorly-controlled asthma. Wheezing on auscultation is concerning, particularly if the wheezing is noticed in phases of the respiratory cycle other than end-expiration.[21]

6. Investigations

Laboratory tests are not routinely required. However, in more severe disease a room air arterial blood gas may be useful in determining baseline oxygenation, carbon dioxide retention, and acid-base status. Pre-operative clinics nearly universally have pulse-oximetry available, which can serve as reasonable surrogate for arterial blood gas in determining baseline oxygenation. A chest x-ray may be obtained to assess for lung hyperinflation and air-trapping. Peak flow measurements are recommended by the American Lung Association for disease self-monitoring and are easily performed at bedside. The suggested "zones" (green=80% or greater than usual, yellow=50-80% of usual, red <50% of usual) alert patients to their current respiratory status.[12] Spirometric tests can be ordered to assess the forced expiratory volume (FEV1), which reflects the degree of airway obstruction. The forced oscillation technique is an emerging tool for assessing bronchial obstruction and reactivity. It seems particularly useful in children or other patients who may not be able to actively participate in spirometry.[23] Recently, the fraction of expired nitric oxide (FeNO) has been evaluated as measure of asthma control.[24] Further study is necessary determine how FeNO should be used along with clinical and spirometric evaluation for managing asthma.[25]Objective testing, physical exam and a careful history need to be synthesized into an overall picture of the patient's current level of disease severity and control so that they can be effectively managed perioperatively.

6.1The Anesthetic Plan

The anaesthetic plan should balance suppression and avoidance of bronchospasm with the usual goals of patient safety, comfort, and a quiet surgical field. Choice of anaesthetic method must be tailored to the patient, the procedure, clinical assessment and the preferences of all involved. No definitive evidence shows that one method is generally superior to another. It seems prudent to avoid direct instrumentation of the airway if at all possible, but anxiety or pain during regional anaesthesia (peripheral nerve or neuraxial block) could themselves precipitate an attack of bronchospasm. Clearly, the highest risk cases are those in which the airway itself is the subject of surgery, or surgery involving the thorax or upper abdomen where tracheal intubation cannot be avoided.[12]

6.2 Preoperative preparation

According to Enright[26], preoperative management in asthmatics should include the following measures:

i. Bronchospasm should be treated with inhaled β2-agonists.
ii. If a patient is at risk for complications, preoperative treatment with 40-60 mg of prednisoneper day or hydrocortisone 100 mg every 8 hour intravenously is suggested. Anyone with apreoperative FEV1<80% of predicted should receive steroids.
iii. Infections should be eradicated using antibiotics.
iv. Fluid and electrolyte imbalances should be corrected, given that high dose β2-agonists cancause hypokalemia, hyperglycemia and hypomagnesemia. In addition to that imbalance, there may be a decreased response to β2-agonists and predisposition to cardiac arrhythmias.
v. Prophylactic cromolyn treatment to prevent degranulation of mast cells and release of mediators should be continued.
vi. Chest physiotherapy improves sputum clearance and bronchial drainage.
vii. Other conditions such as cor pulmonale should be treated.
viii. The patient should stop smoking at least two months before surgery in order to reduce carboxyhemoglobin levels and recovery of endobronchial cilial mucus clearance.

6.3Premedication

An optimal premedication allays anxiety, improves work of breathing, and possibly averts the induction of bronchospasm, while eschewing over sedation and respiratory depression. No ideal drug or drug combination exists for this. The α-2 agonist dexmedetomidine has a favourable profile, including anxiolysis, sympatholysis, and drying of secretions without respiratory depression. By drying secretions and suppressing upper airway vagal responses, anticholinergic agents such as atropine or glycopyrrolate can decrease airway reactivity and should be considered.[12]

6.4 Monitoring

Choice of monitoring should be geared towards assessment of airway mechanics (volumes, pressures, airway flows, I:E ratio, compliance, respiratory waveforms if available). It is very useful to augment the end-tidal CO2 monitoring with a visible waveform so that flattening of the capnogram can be used as an index of expiratory airway flow. However, as bronchospasm worsens, the End tidal CO2 to PaCO2 gradient widens. Consideration should always be given to placing an arterial line in high-risk cases to facilitate ABG measurement.[12]

6.5 Intraoperative management

The overriding goal in anesthetizing an asthmatic patient is to avoid bronchospasm and reduce the response to tracheal intubation. Severe bronchospasm may cause fatal or near-fatal events such as irreversible brain damage due to inability to ventilate.[27] It is extremely important that the patient be at a deep level of anesthesia prior to instrumenting the airway, as tracheal intubation during light levels of anesthesia can precipitate bronchospasm. Regional anesthetic techniques should be considered when appropriate; to avoid airway instrumentation.[28] Intravenous lidocaine has been successfully used to decrease airway irritability.[29] Propofol is the induction agent of choice in the hemodynamically stable patient due to its ability to attenuate the bronchospastic response to intubation both in asthmatics and non-asthmatics.Thiopental or etomidate may also be used as induction agents but lack the bronchodilating properties of propofol and in the case of thiopental, may lead to detrimental histamine release.[30] Ketamine is an ideal induction agent for hemodynamically unstable asthmatics due to its ability to produce direct smooth muscle relaxation and bronchodilation without decreasing arterial pressure or systemic vascular resistance.[31]

Volatile anesthetics are excellent choice for general anesthesia as they depress reflexes and produce direct bronchodilation. Inadequate depth of anaesthesia at any point can allow bronchospasm to be precipitated. Anaesthetic maintenance with a volatile agent such as isoflurane or sevoflurane confers protective bronchodilation. However, there is evidence that desflurane provokes bronchoconstriction in smokers. Halothane has been favoured in the past, but now is not as available, is more blood-soluble leading to longer induction times, and in the setting of hypoxaemia or acidaemia could potentiate arrhythmias.[12]

Warm, humidified gases should be provided at all times. Rapid sequence or standard induction should be performed as indicated as long as adequate anaesthesia is assured.[12]

Succinylcholine, which releases low levels of histamine, has been used safely in asthmatics with little morbidity.[21] Depending on which type of muscarinic receptor is stimulated, increased or decreased bronchial tone and reactivity can be expected. It has been shown that muscle relaxants which affect M2 receptors more than M3 receptors (gallamin, pipecuronium, and rapacuronium) can cause and enhance bronchoconstriction. Otherwise, muscle relaxants which seem to bind M3 receptors more or at least the same way as M2 receptors do not induce bronchospasm. Among those, vecuronium, rocuronium, cisatracurium, and pancuronium are considered safe.[26] In addition to these direct effects on muscarinic receptors, atracurium and mivacurium dose dependently release histamine and have been

identified as triggers of bronchoconstriction and should be used carefully in asthmatic patients.[22]

The decision whether to intubate the trachea, provide anaesthesia by mask, or use a laryngeal mask airway (LMA) is a clinical one. However, there is evidence that tracheal intubation causes reversible increases in airway resistance not observed with placement of an LMA.[12]

In selecting a ventilatory mode, attention should be given to providing an adequately long expiratory time to avoid the build-up of intrinsic or auto-PEEP. This can be facilitated by using higher inspiratory flow rates or smaller tidal volumes than usual. Patients should be kept adequately hydrated as usual, but fluid overload, pulmonary congestion, and oedema can precipitate bronchospasm ('cardiac asthma').[12]

6.6 Acute intraoperative bronchospasm (Perioperative Bronchospasm)

Signs of airway obstruction consistent with bronchospasm include elevation of the peak inspiratory pressure, prolonged expiratory phase, and visible slowing or lack of chest fall.[12] Capnography, shows narrowed airways and prolonged expiration result in a delayed rise in end-tidal Carbon dioxide, producing a characteristic 'shark-fin' appearance.[32]

The chest (and, if not accessible, the expiratory limb of the anaesthetic circuit) should be auscultated to confirm wheezing. Diminished or absent breath sounds can be an ominous sign suggesting critically low airflow. The differential diagnosis includes mucous plugging of the artificial or native airway and pulmonary oedema. A unilateral wheeze suggests the possibility of endobronchial intubation, foreign body obstruction such as a dislodged tooth, or even a tension pneumothorax. If none of these conditions exists, or if bronchospasm persists after they have been corrected, a treatment algorithm for acute intraoperative bronchospasm should be instituted.[12]

7. Treating Perioperative Bronchospasm

The aims of treatment are to relieve airflow obstruction and subsequent hypoxemia as quickly as possible. When isolated perioperative bronchospasm occurs, oxygen concentration should be increased to 100%, and manual bag ventilation immediately started to evaluate pulmonary compliance and to identify all causes of high-circuit pressure.[33] Increased concentration of a volatile anesthetic (sevoflurane, isoflurane) is often useful with the exception of desflurane because of its airway irritant effects, particularly in smokers.[34]

Deepening anesthesia with an intravenous anesthetic (propofol) may be required because intubation induced bronchospasm may be related to an inadequate depth of anesthesia. Inhaled short-acting β2-selective agents (mainly using terbutaline and salbutamol) are key drugs for the fast relief of bronchoconstriction. Their onset of action occurs within 5 min, peak effect is within 60 min and duration of action is 4–6 hr. They should be immediately administered via a nebulizer (8–10 puffs to achieve appropriate therapeutic levels, may be repeated at 15- to 30-min intervals) or, if available, with a metered-dose inhaler (5–10 mg/h) connected to the inspiratory limb of the ventilator circuit. There is no difference in efficacy between terbutaline and salbutamol. Continuous rather than intermittent administration of salbutamol results in greater improvement in PEF and FEV1. Moreover, nebulized epinephrine has no beneficial effect compared with terbutaline or salbutamol.[6]

Systemic glucocorticosteroids should not be omitted. Parenteral steroids also remain a key drug in the treatment of bronchospasm because they speed resolution of exacerbations by decreasing airway inflammation. Systemic glucocorticosteroids such as methylprednisone (1 mg / kg) are preferred over cortisone because their antiinflammatory effect is more potent. However, the antiinflammatory benefit is not immediate.[6]

Combined nebulized ipratropium bromide with a nebulized β2-agonist produces greater bronchodilatation than a β2-agonist alone. Magnesium plays a beneficial role in the treatment of asthma through bronchial smooth muscle relaxation, leading to the use of intravenous or inhaled preparations in patients with severe bronchospasm that fails to be relieved with β2-agonists.

Accordingly, salbutamol administered in isotonic magnesium sulfate provides greater benefit when compared with that diluted with saline.[35]

Systemic epinephrine currently, no recommendations regarding epinephrine can be proposed, except that its use would be reasonable as a rescue therapy in patients with severe asthma complicated by hypotension.[6]

7.1 Emergence and postoperative care

Laryngospasm, bronchospasm and hypoxaemia are major hazards of the emergence phase. Suctioning of the airways must be rendered cautiously. Aspiration also triggers severe bronchospasm. Prophylactic use of anti-emetic agents, antacids and gastric suctioning before emergence should be considered. Respiratory

mechanics must be fully assessed before extubation and emergence.[12]

If acute bronchospasm persists at the end of the case or if it has been severe, or if the patient has a difficult airway, trauma, or a full stomach, consideration should be given to a period of postoperative mechanical ventilation to avoid having to reverse neuromuscular block and to allow time for airway recovery. Repeat administration of a β2-agonist such as albuterol before emergence is advised. When emergence does occur, it should be with adequate analgesia in place, whether intravenous or neuraxial. Dexmedetomidine can be a useful ancillary agent for the reasons already discussed. Reversal of neuromuscular block has a number of hazards. Neostigmine increases bronchospasm risk because of its muscarinic and pro-secretory effects.[36] These can be blunted by coadministration of atropine or glycopyrrolate,but the duration of action of neostigmine can outlast that of the vagolytic agent, especially in the presence of renal insufficiency.[12] Sugamedex is novel agent but availability is the problem.[21]

'Deep extubation' (tracheal extubation while still deeply anaesthetized) has been practiced for many years, especially in children, but it has its own inherent hazards. It mandates full reversal of neuromuscular block. Even if tracheal extubation is smooth, emergence through the arousal stage can initiate severe bronchospasm with an unprotected airway. The risk of regurgitation and aspiration is ever-present.[12]

The keys to minimizing postoperative pulmonary complications are vigilance for bronchospasm and its causes; good pain control, be it by the neuraxial route or patient controlled analgesia[37];bronchodilator therapy; incentive spirometry, deep breathing exercises, and early mobilization.[38]Control of gastro-oesophageal reflux is beneficial in asthma.[39] Non-invasive positive pressure ventilation is an option in some asthmatics that have persistent bronchospasm after tracheal extubation.[40]

8. Conclusion

Good understandings of etiopathogenesis, early diagnosis, and better medical care have resulted in decreasing mortality and morbidity related to bronchial asthma. Though the incidence of bronchial asthma is on the rise, the incidence of perioperative bronchospasm is relatively low in asthmatics undergoing anaesthesia, but when it does occur it becomes an anesthetic hazard. The use of stepwise approach to its diagnosis and intervention would lead to earlier recognition and better management. The keys are detailed preoperative assessment, pharmacotherapy, safe anaesthetic measures throughout peri and postoperative period.

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