BMH MEDICAL JOURNAL

BMH Med. J. 2018;5(4):98-103 **Review Article**

Bronchospasm Under General Anaesthesia

Rajesh MC

Baby Memorial Hospital, Kozhikode 673004

Address for Correspondence: Dr. MC Rajesh MD, MBA, Senior Consultant Anaesthesiologist, Baby Memorial Hospital, Kozhikode, Kerala, India. PIN: 673004. E- mail: rithraj2@yahoo.co.in

Introduction

Intraoperative bronchospasm caused by the spasmodic contraction of bronchial smooth muscle is a rare but potentially lethal challenge for anaesthesiologists. Retrospective analysis shows an incidence of 1.7 per 1000 patients with a confirmed higher possibility in the presence of irritable airway pathology [1] and accounts for almost similar percentage of ASA closed claims study [2]. It can happen in isolation or as a part of more serious pathology such as type E immunoglobulin mediated anaphylaxis.

Bronchospasm results in decreased airway calibre and altered distribution of gases within the lungs leading to ventilation perfusion mismatch. Patients with reactive airway diseases should be considered a potentially at risk candidates for peri operative bronchospasm with increased chance of peri-operative mortality and morbidity if not adequately controlled.

The goal of anaesthesiologist for at risk patients should be to administer safe and sufficient anaesthesia without precipitating bronchospasm. The anaesthesiologist should be wise in choosing proper techniques and avoiding drugs which can precipitate an intraoperative bronchospasm. Judicious use of anti-inflammatory and bronchodilatory medications should be instituted as part of preoperative preparation so that patients are asymptomatic while taking up for procedure in an elective case [3]. It is important to extend the care to the post operative settings also. Good analgesia, post operative respiratory monitoring and educating patients with proper breathing exercises are important to prevent peri-operataive respiratory morbidities.

Who are the patients at risk?

A definite history of obstructive pulmonary disease may not be evident in all patients with bronchospasm. Also most patients with obstructive pulmonary disease may not develop bronchospasm under anaesthesia. Though the incidence of severe peri operative bronchospasm is rare in severe asthmatics, when it does occur, it can be life threatening [3]. There are certain situations considered to be at risk for intra operative bronchospasm due to underlying bronchial hyperactivity. This include recent viral upper respiratory tract infections, exposure to tobacco smoke, history of atopy and recent hospital admission for exacerbations of bronchial asthma [5]. Of course airway instrumentation is a potential stimulus which can trigger bronchoconstriction in patients with irritable airways [4]. Kim ES et al has shown that unlike laryngeal mask airway insertion,

endobroncheal intubation produces reversible bronchoconstriction [6]. Manipulation of the airway or surgical stimulation under light anaesthesia increases the risk of bronchospasm. Airway soiling due to secretions, regurgitation or aspiration in a patient with irritable airway state is a potential stimulus for bronchospasm. An analysis of incidence reported showed the principal causes of bronchospasm as airway irritation (35%) followed by problems with endotracheal tube (23%) and aspiration of gastric contents (14%) [8].

Rule out mechanical causes of increased peak airway pressure

Obstruction of natural or artificial air passages like kinked endotracheal tube, breathing circuit or air filters, smaller endotracheal tubes, overinflation of the tracheal tube cuff, patients active respiratory efforts and fighting with ventilator, endo bronchial intubation are commonly missed causes for increased peak airway pressure. These possibilities should be ruled out and corrected first. An excessive tidal volume or high inspiratory flow rates set in the anaesthesia machine, opioid induced muscle rigidity, also will results in a high peak airway pressures in the monitor suspecting a bronchospasm. Steep head down position, excessive pneumoperitonium, morbidly obese patients are conditions contributing to excessive peak airway pressures during IPPV.

Other than as a separate entity, bronchospasm could be associated with life threatening conditions which requires immediate action like anaphylaxis, pulmonary aspiration, pneumothorax, pulmonary edema and pulmonary embolus.

How to diagnose?

Suspicion is the key to early detection. Under anaesthesia, common presenting signs are expiratory wheeze on auscultation over the chest or heard in the breathing circuit, decreased pulmonary compliance and falling oxygen saturation. Spontaneously breathing patient will show prolonged exhalation. To demonstrate a wheeze, it requires some movement of air. So in severe cases can expect even a silent chest on auscultation and correct diagnosis in such circumstances is made by the increased inflation pressures. Similarly breath sounds may be uniformly reduced also. On positive pressure ventilation, peak airway pressure is increased, tidal volume reduced or both. Narrowed airways and prolonged expiration results in a characteristic shark fin appearance in the capnography wave form (Slowly increasing wave on the capnography) and decreasing tidal volume. Positive pressures when delivered before the expiratory phase can result in the development of intrinsic positive end expiratory pressure. This auto/intrinsic PEEP can result in an increase of intra thoracic pressure, decrease venous return and impair cardiac output.

When bronchospasm is associated with anaphylaxis or allergy, the signs exhibited by the patient include rashes and hypotension in addition to increased inflation pressure, fall in saturation and capnography changes.

Implication of Pharmacological agents

Pharmacological agents which can potentially precipitate a bronchospasm has to be kept in mind while anaesthetising a high risk patient. These include beta adrenergic antagonists, acetylcholinesterase inhibitors, NSAIDs. Non specific beta blockers should be best avoided. But ultra short acting highly selective beta-1 antagonists like esmolol and landiolol are used peri operatively without much problem. Use of acetylcholinesterase inhibitors for neuromuscular blockade reversal should be combined with adequate doses of anticholinergic agents. The concern with acetylcholinesterase inhibitors is because of its muscarinic side effects. But in practice this is not a major problem. Sugammadex which encapsulates steroidal neuromuscular blocking agents without muscarinic side effects is proposed as an alternative to regular anticholinesterase inhibitors. There are some who advocate avoiding reversal altogether allowing adequate time for the recovery of neuromuscular function. Certain volatile anaesthetic agents like Isoflurane and Desflurane when

introduced quickly can trigger bronchospasm. Drugs with the potential for histamine release like thiopentone, atracurium, morphine, mivacurium, protamine can also precipitate bronchospasm. Sometimes Fentanyl induced muscle rigidity can grossly reduce the chest wall compliance increasing peak airway pressure to mimic bronchospasm.

Perioperative care in high risk patients

Whenever possible, regional anaesthesia should be preferred if the site of surgery is suitable since it reduces airway manipulations. If a general anaesthesia is perceived to be mandatory, supraglottic airways to be preferred over routine endotracheal intubations. In the event of emergency surgery in asthmatic patients, anaesthesiologists have to balance the risk between protecting the airway from risk of aspiration and the risk of triggering bronchospasm. Scaltaro P et al has shown that the increase in airway resistance that can occur after tracheal intubation can be reduced by prophylactic administration of bronchodilators [7]. In fact, airway irritation at the time of intubation is a known potential cause for bronchospasm. According to Westhorpe RN et al, bronchospasm is most commonly encountered during induction and maintenance than at the time of emergence and recovery phase [8]. So it is important to make the patient at a deeper plane prior to instrumentation of airway. Use of intravenous Lidocaine because of its membrane stabilizing effect is found to be useful to blunt the airway irritability during airway instrumentation [9]. It has to be administered intravenously 60 to 90 seconds before intubation (1 to 1.5 mg/kg body weight). Similarly extubation can also be performed under deep planes.

Asthmatic patient with wheeze, irritable cough,recent respiratory infections, shortness of breath, recent exacerbations or hospital admission indicate poor control. Patients should be asked to continue their medications with which they are having symptom control. Children with active upper respiratory obstruction may necessitate postponing surgery till resolution of symptoms [3]. Asthmatic children requires pre operative optimisation before any elective procedure. For a well controlled asthmatic, use of inhaled beta-2 adrenergic agonists 1 to 2 hours before surgery may be enough [10]. An asymptomatic or moderately controlled asthmatic requires additional optimisation with inhaled anti inflammatory agents along with regular use of nebulised adrenergic beta-2 agonists [10]. Whereas a poorly controlled asthma requires in addition to beta-2 agonists and inhaled steroids systemic steroids. Either oral prednisolone (1mg/kg/day maximum of 60 mg) 3 to 5 days before surgery or dexamethasone (0.6 mg/kg maximum of 16 mg) can also be used [11].

Smokers should be encouraged to stop smoking preoperatively. It was observed that those who quit smoking more than four weeks before surgery have lower risk of pulmonary complications compared to those who continued to smoke [12].

Adequate pain relief, hydration, correction of electrolyte imbalance, anxiolysis, humidification of inspired gases, airway suctioning under deep planes of anaesthesia are some of the general measures to be given importance to prevent a peri operative event.

Propofol is an ideal agent for induction in an haemodynamically stable patient because of its ability to attenuate bronchospastic response to airway instrumentation [13]. For maintanence of anaesthesia, Sevoflurane is preferred because of its bronchodilatory effects [14].

Ventilator strategies should be altered to limit the peak inspiratory pressure, tidal volume and lengthening the I:E ratio to avoid auto PEEP.

Intra operative crisis

Suspected bronchospasm should be assessed and treated promptly. Management starts with

ventilating the patient with 100% oxygen and calling for help early. Manual bag ventilation should be started immediately to assess the compliance.

It is important to rule out mechanical causes of airway obstruction and inadequate plane of anaesthesia before initiating treatment. The asthma related bronchospasm usually respond to deepening of anaesthesia with volatile anaesthetic agents if haemodynamic status permits. But in severe cases effective delivery of inhalational agents will be difficult. Intravenous agents like Propofol are an ideal alternative in such situations to deepen the plane. Ketamine on the other hand produces bronchodilatation and is a widely available agent. If bronchospasm still persists, then institution of beta-2 agonist therapy should be considered [15]. Short acting metered dose inhaler is the corner stone of management of intra operative bronchospasm. With the advent of more specific agents (Salbutamol, Terbutaline) the problem related to tachyarrythmias of older agents never come to the picture. Only a small fraction of the inhaled agent is absorbed systemically. Metered dose inhaler (MDI) delivery should synchronize with the onset of inspiratory flow and heat and moisture exchanger (HME) should be removed during delivery of the drug. MDI should be delivered to the inspiratory limb of the circuit. Delivering drugs through endotracheal tube is a challenge because majority of drug is lost in the endotracheal tube. So a relatively large dose like 8 to 10 puffs should be given. If in spite of beta-2 agonist therapy and deep general anaesthesia, bronchospasm is not relieved, steroid administration may be necessary (methyl prednisolone 125 milligram I/V). But for the therapeutic benefits of corticosteroids to become apparent, it will take several hours (4-6 hours). Anticholinergics is an accepted bronchodilating agent. There is evidence for the addition of Magnesium Sulphate for asthma [16] (50 mg/kg IV over 20 minutes, maximum 2 gram). Ipratopium bromide 0.5 mg nebulised 6 hourly in combination with beta-2 agonists produce maximum bronchodilatation. Ketamine bolus 10 to 20 mg or infusion 1-3mg/kg/hr and in extreme cases Epinephrine (Adrenaline) nebulised 5 ml 1:1000, intravenous 10 microgram (0.1ml 1:10000) to 100 microgram (1ml 1:10000) titrated to response have been tried. Use of Epinephrine is best reserved in intractable cases especially in the presence of hypotension.

Nitroglycerin because of its direct smooth muscle relaxation is found to have a positive effect in severe bronchospasm [17]. Helium oxygen mixture (Heliox) have been used to provide laminar flow during bronchospasm. The theoretical advantage with Heliox is the ability to provide ventilation till corticosteroids take effect. But to what extend this can be incorporated in the protocol can't be commented at present because of insufficient data [18].

Recently an analogue of Lidocaine, known as Lidocaine JM25-1 was pharmacologically evaluated [19]. It was found to have less of local anaesthetic action because of limited impact on the sodium channels but more effective as a bronchodilator and anti-inflammatory agent [19, 20].

Management algorithm should also include frequent blood gas assessment to know the extent of hypoxaemia and hypercarbia.

Conclusion

Intraoperative bronchospasm is usually triggered by mechanical or pharmacological causes or as a part of a systemic reason like anaphylaxis. For asymptomatic asthmatics the incidence is very low. It is important to identify patients at risk of perioperative bronchospasm and avoid any intra operative insult. Rational use of techniques and agents is the key. Inhaled and parentral beta-2 agonist is the key element in pharmacological therapy along with the judicious use of steroids. Agents like Nitroglycerin, Heliox, and JM25-1 requires more evaluation and research before acceptance in standard treatment protocols.

References

1. Olsson Gl. Bronchospasm during anaesthesia. A computer aided incidence study of 136929

patients. Acta Anesthesiol. 1987;987:244-52.

2. Capla RA, Posner KL, Ward RJ, Cheney FW. Adverse respiratory events in anaesthesia: A closed claims analysis. Anaesthesiol. 1990;72:828-33.

3. Rajesh MC. Anaesthesia for children with bronchial asthma and respiratory infections. Indian J Anaesth 2015;59:584-8.

4. Woods BD, Sladen RN. Peri-operative considerations for the patient with asthma and bronchospasm. BJA 2009;103: i57-i65.

5 Benca J. Bronchospasm. Complications in anaesthesia. Second Edition 2007. 189-92.

6. Kim ES, Bishop MJ. Endobroncheal intubation, but not laryngeal mask airway insertion, produces reversible bronchoconstriction. Anaesthesiology. 1999;90:391-4.

7 .Scaltaro P, Sly PD, Sims C, Habre W. Salbutamol prevents the increase in respiratory resistance caused by tracheal intubation during sevoflurane anaesthetic in asthmatic children. Anaesth Analg 2001; 93:898-902.

8. Westhorpe RN, Ludbrook GL, Helps SC. Crisis management during anaesthesia:bronchospasm. Qual saf health care 20055:e7.

9. Adamzik M, Groeben H, Farahani R, Lehmann N, Peters J. Intravenous Lidocaine after tracheal intubation mitigates bronchoconstriction in patients with asthma. Anesth Analg 2007; 104:168-72.

10. Lauer R, Vadi M, Mason L. Anaesthetic management of the child with co-existing pulmonary disease. Br J Anaesth 2012;109 Suppl 1:i47-59.

11. Liccardi G, Salzillo A, De Blasio F, D'Amato G. Control of asthma for reducing the risk of bronchospasm in asthmatics undergoing general anesthesia and/or intravascular administration of radiographic contrast media. Curr Med Res Opin 2009; 25:1621-30.

12. Wong J,Lam DP, Abrishami A et al. Short term preoperative smoking cessation and post operative complications: a systemic review and meta analysis. Canadian journal of Anaesthesia 2012;9:268-79.

13. Wu RS,Wu KC,Sum DC,Bishop MJ.Comparative effects of Thiopentoneand Propofol on repiratory resistance after tracheal intubation. Br J Anaesth 1996;77:735-8.

14. Rooke GA, Choi JH, Bishop MJ. The effect of Isoflurane, Halothane, Sevoflurane and Thiopental/Nitrous oxide on respiratory system resistance after tracheal intubation. Anaesthesiology. 1997;86:1294-9.

15. Applegate R, Ryan Lauer, John Lenart, Jason Gatling etal. The perioperative Management of Asthma. J Aller Ther 2013; S11:007.

16. Shan Z, Rong Y, Yang W, Wang D, Yao P et al. Intravenous and nebulised magnesium sulphate for treating acute asthma in adults and children:a systemic review and meta analysis. Respir Med 2013;107:321-30.

17. Mikulandra, Simon. Unusual use of Nitroglycerin for severe Bronchospasm during coiling

emboliszation of ruptured Intracranial Aneurysm. Journal of Neurosurgical Anaesthesiology 2008;20:207-8.

18. Linck SL. Use of heliox for intraoperative bronchospasm:a case report. AANA J 2007;75:189-92.

19. Rajesh MC. Lidocaine-Pharmacological actions beyond local anaesthetic. BMH Med J 2017;4:27-30.

20. Magda F, Serra, Josiane S, Neves, Gina C et al. JM25-1, a Lidocaine Analog combining airway relaxant and anti-inflammatory properties. Anesthesiology 2016;124:109-20.