

Reactive airway and anaesthesia: challenge to the anaesthetist and the way forward

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Abstract

Background: Patients with concurrent medical conditions such as Reactive airway disease presenting for anaesthesia, and surgery have potentially increased risk of perioperative morbidity and mortality if not well managed.

Objective: To highlight the need for adequate perioperative care and review the evidence for selection of techniques in the anaesthesia for such cases”

Materials and methods: An illustrative case is presented.

Conclusion: The main goal of the anaesthetist is to administer safe and sufficient anaesthesia without precipitating bronchospasm.

Keyword: Reactive airway, anaesthesia, presentation, management and constraints.

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Introduction

Patients presenting for anaesthesia and surgery may have with co-existing medical conditions¹⁻⁴. Bronchial asthma is one of such diseases¹. The co-existence of any medical condition in the surgical patient has the potential to increase the risk of morbidity and mortality in the perioperative period, if inadequately managed¹⁻⁶. The main aim of this article is to highlight this, and streamline the steps necessary to be taken in the anaesthetic management of patients with such condition. A case of an asthmatic patient with prostatic hypertrophy requiring anaesthesia and surgical resection is presented.

Case Report

A 60year old man presented with complaints of frequent urination, urgency and poor urinary stream for three months. There was no haematuria, dysuria or urethral discharge. He was diagnosed as asthmatic about twenty years before presentation. He was allergic to cold, smoke and dusts. The disease was brought under control using salbutamol tablets 8mg 6 hourly and salbutamol inhaler when required. He was admitted on three occasions because of severe asthmatic attacks and was treated with intravenous fluids, antibiotics, aminophylline, steroids and other drugs he could not recall. His last attack was about

two months prior to presentation. There was no history of cough and had been able to maintain his normal daily activities up to the time he presented.

On examination, he was clinically stable not in respiratory distress. Air entry was vesicular. The cardiovascular system was stable with pulse rate of 84 beats per minute and blood pressure of 150/90mmHg. There were no lesions on any part of the vertebral spine. Rectal examination revealed a palpably enlarged prostate. A diagnosis of benign prostatic hypertrophy in a known asthmatic (controlled) was made. He was scheduled for prostatectomy.

Blood biochemistry and haematological tests were normal. Chest X-ray and E.C.G were all normal. Three units of blood were screened, grouped and cross-matched. He was assessed as ASA III and graded Mallampatti II. He was consented for epidural anaesthesia.

While in the theatre, emergency drugs including aminophylline, hydrocortisone, ephedrine, etc were kept within reach. A vein was cannulated with size 18G cannula and a litre of normal saline set up.

The patient was positioned sitting on the operation table with his feet supported on a stool and hands on the opposite shoulders. The fourth lumbar space (L4/L5 inter-space) was located and infiltrated with 1.0ml of 1.0% plain lignocaine. A size 18G Tuohy epidural needle was inserted with the bevel facing upward. On piercing the interspinous ligament, the stylet was removed and a 20mls resistance-free glass syringe half full of air, attached to the epidural needle. A gentle continuous pressure was applied to the plunger as the needle was

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advanced. A give and later a sudden loss of resistance to the plunger was felt as the epidural space was entered. The glass syringe was removed and a syringe containing 15mls of 0.5% plain bupivacaine connected to the extradural needle. A test dose of 5mls of the solution was administered after aspiration. Five minutes later, the remaining solution was slowly administered while verbal contact was maintained with the patient. The needle, together with the syringe, was removed and small gauze plastered to the site. He was then positioned supine with a 10° head down tilt. After about 15minutes, the Bromage scale was used to assess the extent of the block. The patient could neither raise his legs nor flex his knees or ankle joints. This confirmed the block to be up to the level of the umbilicus (corresponds to T₁₀).

Intra-operative course was uneventful. The pulse rate and blood pressure were fairly stable. The respiratory rate and air entry were frequently checked. Arterial oxygen saturation was monitored by using pulse oximeter. There were no episodes of coughs or secretions. The operation lasted 2hours 15minutes. Post operatively the patient was closely monitored in the recovery room. Oxygen saturation remained good while on room air. He was discharged to the ward after 30minutes of observation in the recovery.

Discussion

A mild tonic constriction exists in all normal human airways. This is mainly maintained by the efferent vagal activity and easily abolished by antimuscarinic drugs like atropine. In some individuals, intense bronchoconstriction is provoked following airway stimulation with low level of stimulus as compared to normal persons. This bronchial hyper-responsiveness is termed "reactive airways". The commonest disease condition that falls into this entity is bronchial asthma. Others include chronic bronchitis, emphysema, allergic rhinitis and upper/lower respiratory tract infections¹. This category of patients constitutes a problem to the anaesthetist especially when the airway has to be tempered with.

Preoperative identification of patients with reactive airways is important in planning a rational approach for proper anaesthetic care. So far, there is no single best test available for identifying or evaluating airway hyper-responsiveness. Often, precise testing is not practicable preoperatively and clinicians have to rely on history to identify factors suggesting an increased likelihood for perioperative bronchospasm. One of the most important is history of recent upper respiratory tract infection characterized by cough and fever. Respiratory symptoms like nocturnal dyspnoea, chest tightness on awakening, associated breathlessness and wheezing in response to various respiratory irritants, appear highly predictive of increased bronchial

reactivity. The case being presented is a diagnosed case of controlled bronchial asthma requiring surgery on the perineum.

Anaesthetizing patients with reactive airway disease is a challenge to the anaesthetist; he has to be selective in the choice of technique and the use of drugs on such patients to avoid the provocation of bronchospasm. Such patients could present in controlled or acute state of the disease for either elective or emergency surgery. Patients presenting in controlled state and for elective surgery present less problems. The operation could proceed while precautions need to be taken to prevent bronchospasm. The case being presented falls under this group. In the case of uncontrolled disease coming for elective surgery, the anaesthetist might be compelled to postpone the operation to such a time that would be safe for anaesthesia/surgery. The challenge in compounded when the uncontrolled/untreated patient presents as a surgical emergency. In such a case, the anaesthetist is compelled to anaesthetize the patient using available resources considered safe, within his reach. Sometimes the drugs considered safe might not be available for use when urgently needed. This has been a constraint and a major challenge to the anaesthetist in the management of such patients.

Asthma is a disease of the airways that is characterized by increased responsiveness of the tracheobronchial tree to multiple stimuli². It is manifested by widespread narrowing of the air passages and may be relieved spontaneously or as a result of therapy^{3,4,5}. The decreased airway caliber associated with bronchoconstriction affects the distribution of gases within the lungs. The major effect of this is under ventilation of many lung units leading to low ventilation perfusion ratio. This results in arterial hypoxaemia predisposing the patient to non-specific bronchial reactivity⁶. This calls for the need to administer high concentration of oxygen in the perioperative management of such patients. Oxygen was administered by face mask to our patient up to the early postoperative period. Drugs that could be needed in the treatment of acute asthmatic attack had to be made available before anaesthesia commences. Such drugs were available in the theatre and within reach before anaesthesia started for our patient.

In the case being presented established diagnosis of asthma had already been made. The disease condition is also under controlled. The challenge to us was in the choice of technique and the use of drugs/equipment free of provoking bronchospasm.

Options available included the use of general anaesthetics devoid of stimulating the airways or employing the use of regional anaesthesia. Being

a perineal surgical case, epidural anaesthesia was chosen for the patient. This eliminates the need for instrumentation and drugs administration, including anaesthetic gases that could trigger bronchospasm^{7, 8}.

Olsson⁹ in a retrospective analysis of bronchospasm during anaesthesia, found an incidence of 1.7 per 1000 patients (1 case per 634 anaesthetics). He also confirmed a higher incidence in the presence of pre-existing airway obstruction, especially during airway instrumentation. Avoiding the airways help in preventing of bronchospasm in our patient. Intraoperative bronchospasm is diagnosed by ventilatory difficulties characterized by increased peak airway pressure and expiratory wheezing. The key to therapy is inhalation of sympathomimetics such as albuterol. These drugs produce more rapid and effective bronchodilation than intravenous aminophylline. Intravenous lignocaine (1.5-2 mg/kg), hydrocortisone (4 mg/kg) or glycopyrrolate (1 mg) may also help in reversing the reflex response to bronchoconstriction. Glycopyrrolate may also be administered directly through the endotracheal tube. These drugs are likely to be more effective before the stimulus. Lignocaine has been shown to prevent such bronchospasm by blockade of the airway response to irritation¹⁰ and direct attenuation of smooth muscle response to irritation¹¹. Its use could therefore be employed when endotracheal intubation and some drugs likely to trigger bronchospasm are indicated. Inhalational anaesthetic agents like halothane produce bronchodilatation and appear to prevent the development of bronchospasm¹². If inhalation anaesthesia is indicated, halothane has been considered the agent of choice; but its myocardial depressant and arrhythmic effects in the presence of catecholamines may limit its use. At high MAC (>1.5), both enflurane and isoflurane prevent vagally mediated bronchospasm¹³. These agents could hence be considered useful for inhalation anaesthesia in the asthmatics. However in 1997, Rooke et al¹³ observed clinically in humans that sevoflurane at 1.1 MAC may be more efficacious than both iso- and enflurane. The beta- adrenergic aerosols like salbutamol, when inhaled before induction of anaesthesia prevents bronchospasm¹⁴. Our patient did not suffer any asthmatic attack throughout the perioperative period.

Conclusion

Identification of “at risk patients” and adequate preoperative preparation are essential for the administration of safe anaesthetics to patients with reactive airway disease. Careful selection of anaesthetic technique and drugs will reduce morbidity and mortality during the perioperative period.

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