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Case Report

Crisis Management of Perioperative Bronchospasm in a Neonate with Sepsis

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Abstract: Bronchospasm in association with anaesthesia may appear as an entity in its own right or be a component of another problem such as anaphylaxis. It may present with expiratory wheeze, prolonged exhalation or, in severe cases, complete silence on auscultation. Although most cases are handled appropriately by the attending anaesthetist, the use of a systematic approach to its diagnosis and management would lead to earlier recognition and/or better management of this fatal intraoperative anaesthetic crisis.
Keywords: Bronchospasm; sepsis; bronchial hyperreactivity; salbutamol.

INTRODUCTION

Bronchospasm during general anaesthesia can present as a single isolated entity or as a component of a more serious underlying problem such as anaphylaxis especially after induction/intubation [1]. Untreated it can cause hypoxia, hypotension and increased mortality and morbidity. This case report illustrates the successful management of perioperative severe bronchospasm in a neonate with sepsis scheduled for exploratory lapratomy. Peroperative bronchospasm under GA is relatively uncommon in patients with reactive airway disease (0.2%). Usually patients with COPD, SIRS show hyperreactive airway responses to mechanical and chemical irritants.

CASE REPORT

A 15 days old female neonate (wt 1.4 kg) with jejunal obstruction was posted for emergency exploratorylapratomy. It was an uneventful institutional LSCS due to obstructed labour (G1P1A0) with no significant family history of atopy, or any other chronic diseases. Onauscultation, b/l lung fields were normal with slight wheeze and normal heart sounds. Abdominal distension was present. PR – 168/min SpO2- 94%.

Routine investigations - (Hb—9.5g/dl;TLC-12,600/mm^3; Platelets-0.25 lac/mm^3;Ser. Urea-46.2mg/dl;Ser. Creatinine-0.60mg/dl; Ser. Na^+-132.8mmol/l; Ser K^+-5mmol/l;Ser. Ca^{++}-1.07mmol/l).

Premedication was done with Inj. Atropine 0.04 mg; Inj. Ondansetron0.2 mg; Inj. Fentanyl 2ug IV, preoxygenated with 4 ltr/min of O_2 for 3mins and induction was done with ketamine 3 mg iv and endotracheal intubation with uncuffed ETT 2.5mm size was facilitated by Inj Sch. 3 mg iv. On chest auscultation after intubation b/l wheeze was present, simultaneously saturation began to drop from 98% to 54%. As esophageal intubation was suspected, the patient was immediately extubated and mask ventilation was started. Inj hydrocortisone 5mg ivwas given and saturation started to rise gradually. After 2-3 minutes when spontaneous respiratory movements were appreciated. Injatracurium 1mg was given IV to facilitate reintubation.

Intubation was again attempted by another anaesthetist when saturation reached 100% after mask ventilation followed by worse incidence of desaturation even after under vision intubation and inadequate chest expansion with B/L wheeze. Now endobronchial intubation was suspected so ETT was withdrawn to 0.5 cm but to no avail.Etco2 demonstrated marked prolonged expiratory upstroke of the capnogram.SpO2 continuously dipped again to 55% with bradycardia (50

bpm) which was followed by peripheral cyanosis within<2 min of intubation. Considering severe bronchospasm as the cause, salbutamol inhaler 2-3 puffs were given down the ETT directly. As no improvement was seen, estuation was done and BMV was resumed. Simultaneously CPR was started. Inj atropine 0.03 mg IV and Inj Epinephrine 0.01 ml (1 in 10,000) was administered and patient was resuscitated successfully with the return of normal HR.

Ventilation was continued by bag and mask with increased pressure and high O2 flow rate 6 let/min and maintaining 100% saturation on it. As the patient and vitals were stable, also atracurium (NDMR) had been given and the surgery was an emergency so could not be rescheduled, it was decided by the team to proceed with BMV (no supraglottic device of that size was available) on O_2 and sevoflurane 4% and surgeons were asked to use LA at the incision site. Regular suctioning of riles tube was done intraoperatively to deflate the gut. Surgery ended uneventfully after 40 min. Patient was shifted to NICU after she was completely reversed.

DISCUSSION

a) Bronchospasm- It is characterized by prolonged expiration, wheezes and increased airway pressures during IPPV producing a characteristic 'shark-fin' appearance [1]. Wheezing requires movement of gas through narrowed airways and so in severe bronchospasm wheeze may be quiet or absent. Other causes of wheeze during GA are partial obstruction of tracheal tube (including ETT abutting the carina or endobronchial intubation); Bronchospasm; Pulmonary oedema; Aspiration of gastric contents; Pulmonary embolism; Tension pneumothorax; Foreign body in the tracheobronchial tree. As it is a highly fatal anaesthetic crisis management of the suspected underlying cause must be done promptly.

b) Predisposing factors-Various factors predisposing bronchospasm can be h/o URTI ; allergy ; URTI bronchial hyperreactivity; mechanical (i.e. intubation-induced); pharmacologic-induced (via histamine-releasing drugs such asatracurium or mivacurium, beta blockers, NSAIDs, cholinesterase inhibitors); airway soiling (unexplained bronchospasmespecially in patients without increased risk of airway hyperreactivity, should prompt consideration of airwaysoiling due to secretions, regurgitation or aspiration.it is more common with LMA than ETT) [3]. Certain surgical procedures have highly stimulating stages that can trigger bronchospasm (and laryngospasm). Examples of these include anal or cervical dilatation, stripping of the long saphenous vein during varicose vein surgery and traction on the peritoneum. In our case, it could be airway soiling and also the patient was very low birth weight and in sepsis both of which may act as risk factors for predisposing bronchospasm.

c) Differential diagnosis- This includes inadequate anaesthesia, mucous plugging of the airway, esophageal intubation, kinked or obstructed tube/circuit, and pulmonary aspiration. Unilateral wheezing suggests endobronchial intubation or an obstructed tube by a foreign body (such as a tooth). If the clinical symptoms fail to resolve despite appropriate therapy, other etiologist such as pulmonary enema or pneumothorax should also be considered. Latex-induced anaphylaxis typically occurs in patients with a history of atopy. As latex proteins are slowly absorbed, latex-induced anaphylaxis usually occurs up to 30–60 min after the beginning of surgery. Laryngospasm should be considered and excluded. In non-incubated patient's acute laryngospasm can produce upper airway noise (usually inspiratory), reduced breath sounds and difficulty in ventilation. It can present with signs of airway obstruction including increased respiratory effort, tracheal tug and paradoxical movement of the chest and abdomen ('see-saw' respiration).

d) Management-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. Increased concentration of a volatile anaesthetic (sevoflurane, isoflurane) is often useful with the exception of desflurane because of its airway irritant effects, particularly in smokers. Deepening anaesthesia with an intravenous anaesthetic may be required because intubation- induced bronchospasm may be related to an inadequate depth of anaesthesia as was done in this case.

Short-acting β -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 h. 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.

Other drugs which can be used are ipratropium bromide; magnesium sulphate; ketamine; aminophylline; hydrocortisone; epinephrine.

e)Respiratory morbidity and sepsis-Neonatal sepsis continues to be a common and significant health care burden, especially in very low birth weight infants(VLBW< 1500 g) [4].Clinical signs and symptoms are non-specific include temperature instability, bradycardia and apnea, hypotension. As it is a systemic inflammatory state [5], there may be increased incidence of bronchospasm due to bronchial hyper reactivity.

f) Prevention-Patients with risk factors should be thoroughly assessed and care taken to ensure they are optimised for surgery. Recent or frequent exacerbations or admission to hospital may be an indication to postpone non-essential surgery. Preoperative bronchodilators, inhaled or oral corticosteroids, chest physiotherapy and referral to a respiratory physician may all be appropriate.

g) Postoperative care-With ongoing symptoms a chest radiograph should be requested and reviewed to exclude pulmonary oedema and pneumothorax. Inappropriate, regular therapy (bronchodilators, corticosteroids, chest physiotherapy) should be arranged as secondary management. With ongoing bronchospasm, arrangements should be made for the patient to go to a high dependency or intensive care unit [1].

CONCLUSION

Perioperative bronchospasm may range in presentation from complete silence on auscultation or a few quiet musical notes at the end of exhalation to loud discordant expiratory noise. The plan should not only treat the problem and its pathophysio- logical consequences, but should also prompt the anaesthetist to re-review the patient's evolving condition. Finally, it is important that a full explanation of what happened be given to the patient, that the problem be clearly documented in the anaesthetic record, and that the patient be given a letter to warn future anaesthetists. If a particular precipitating event was significant or a particular action was useful in resolving the crisis, this should be clearly explained and documented [2].

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