Bronchospasm in a Patient with Asthma

INDIANA UNIVERSITY SCHOOL OF MEDICINE

Hannah Armetta SAA, Dominic Ng CAA, Elizabeth Emhardt, MD

Indiana University School of Medicine and Indiana University Health, Indianapolis, Indiana

Indiana University Health

OBJECTIVES

- Review the mechanism of a bronchospasm
- Review ways to avoid a bronchospasm
- Discuss ways to treat a bronchospasm

BACKGROUND

Asthma is a chronic airway disease. In asthmatic patients, a trigger leads to inflammation due to the hyperactivity in the bronchioles causing the bronchial tree to close off. This causes a lower airway obstruction . Of these asthmatic patients 9% have a bronchospasm post induction and 25% have wheezing at some point during the perioperative period

Mechanism of a bronchospasm: irritant at the level of the afferent nerve that triggers a reflex that is sent to the nucleus of the solitary tract which stimulates the vagus nerve which then dumps ach into the lungs where the M3 receptors cause bronchoconstriction.

CASE REPORT

47 year old female presenting for an emergent sigmoidoscopy due to multiple gun shot wounds to the thigh and pelvic. Patient told EMS that she had no medical issues.

Once in the OR the patient stated that she had asthma and used an inhaler at home.

The plan was for general anesthesia with an endotracheal tube.

ASA monitors where placed and the patient was induced with the below medications:

- 100 mcg fentanyl, followed by a second 100 mcg of fentanyl
- 200 mg propfol
- 100 mg succinylcholine
- 100 mg Lidocaine

The induction was uneventful and placement of tube was confirmed.

RESULTS

Following induction 6 mg of decadron and 2 G of ancef were administered. The expiratory sevoflurane shortly after induction was 2.8. The sevoflurane was turned down slightly and to an expiratory sevoflurane level of 2.4. Shortly after the sevoflurane was decreased the ventilator alarmed with unable to reach tidal volumes. The patient was then put on 100% O2, taken off the ventilator and bag ventilation was attempted and successful with some tightness to the bag. Wheezing was heard bilaterally and an obstructive waveform was observed on the ETCO2 tracing. With this information a bronchospasm was diagnosed.

The anesthetic was deepened, with little effect, while albuterol and ketamine were retrieved from pharmacy. 10 puffs of albuterol were administered, with slight decrease of peak pressures. 30 mg of ketamine was administered to the patient, after which the patients peak pressure returned to the normal range.

The sevoflurane was kept at 2.7 for the rest of the case with little effect to blood pressure. The remainder of the case was uneventful. Prior to extubation 20 mg of ketamine was administered along with 10 more puffs of albuterol. Extubation was uneventful and no other respiratory issues occurred.

DISCUSSION

Treatment pathway for a bronchospasm

1. 100% O2
2. Deepen the anesthetic
3. Rule out other potential causes
4. Albuterol +/- inhaled anticholinergic such as ipratropium
5. Also, consider the following
    1. Ketamine
    2. Epinephrine
    3. Hydrocortisone
    4. Nebulized racemic epinephrine

LTA – Laryngotracheal anesthesia, Lidocaine: Lidocaine that is sprayed into the tracheal in order to anesthetize the airway and prevent the communication of irritation up to the solitary tract. These work for 1-2 hours, assisting with blocking any irritation both during and after induction.

Sevoflurane – when a patient is kept deep enough, this inhibits communication between CNS and prevents efferent responses down the vagus nerve. Mechanism for causing vasodilation is not fully understood, it is thought that it reduces intracellular Ca thus being responsible for its bronchodilation

Ketamine: There have been many proposed ways that ketamine works to cause bronchodilation. By blocking NMDA receptors in the lungs it inhibits the NMDA receptor activated airway constriction. Bocks the presynaptic reuptake of norepinephrine leading an increase of norepinephrine at the synapse, these act at the Beta 2 receptors causing bronchodilation. It also has been shown to have an anticholinergic by inhibiting vagal outflow, decreasing Ca influx leading to increased bronchiole relaxation.

Albuterol – The Beta 2 receptor which increases cAMP which inhibits MLCK from being able to initiate contraction and causes relaxation.

Inhaled anticholinergics – work by blocking M2 and M3 receptors. If there is a triggering agent and the vagus nerve releases a large about of ach into the lungs the M2 and M3 receptors will be blocked and there will be limited parasympathetic mediated bronchoconstriction.

Epinephrine – works directly at the B2 receptors in order to increase cAMP to inhibit MLCK from initiating contraction, thus causing relaxation

ALTERNATIVE PLAN

Due to the fact this was an emergent case there was no time for longer term preoperative optimization. With little known at the time of the true severity of the patients asthma it would have been to our benefit to pretreat the patient with albuterol. The patient had stated that they were properly NPO, even though this was a traumatic injury, an LTA could have been used to blunt the airway reflexes triggered at induction. With knowledge of the patient's asthma history it would have been ideal to have albuterol and/or ketamine in the room in order to be prepared for a bronchospasm to occur

CONCLUSIONS

Even though there is no way to guarantee that your asthmatic patient will not have a bronchospasm it is important to take steps along the way to try and decrease the likelihood of a bronchospasm occurring. Preoperative optimization is an important step in bronchospasm prevention. It is also important to have bronchodilators available such as albuterol, inhaled anticholinergics, ketamine, and epinephrine in order to promptly treat the bronchospasm once recognized.

Prompt recognition and treatment of a bronchospasm is vital in the prevention of life threatening hypoxia and circulatory collapse

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