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Case Report

Bronchospasm Under Spinal Anesthesia : A Report of Two Cases, with a Literature Review

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ABSTRACT

Bronchospasm in spinal anesthesia is uncommon. We are reporting two cases of bronchospasm under spinal anesthesia in patients with hyper-reactive airways who had no respiratory signs or symptoms preoperatively.

Case 1: A 50 yr old male, known case of bronchial asthma on treatment underwent emergency fasciotomy for cellulitis on his left leg under spinal anesthesia, after receiving steroid prophylaxis for his asthma. Case 2: A 27 year old male, underwent debridement and k wire fixation of his right medial malleolus under spinal anesthesia. This patient had a history of allergic rhinitis which was not revealed on preanesthetic evaluation.

Both of them received subarachnoid block with 10 mg 0.5% hyperbaric bupivacaine and a block height of T8 and T10 was achieved in case 1 & 2 respectively. In these cases severe bronchospasm (coughing, wheezing, desaturation) occurred 15 min and 5 min after spinal injection in the respective cases. Both were successfully treated with bronchodilators and steroids.

Bronchial asthma and allergic rhinitis share a common immunopathogenesis, with hyper-reactive airways. These patients have a dominant parasympathetic tone which maybe unmasked after sympathetic blockade occurring in spinal anesthesia. This could be the possible mechanism of bronchospasm in the reported cases.

Keywords: bronchospasm, spinal anesthesia, complications, allergic rhinitis, bronchial asthma, hyperactive airways.

INTRODUCTION

Bronchial asthma and allergic rhinitis have a common immunopathogenesis with hyperactive airways and should be considered a single respiratory disease. [1] Regional anesthesia is the preferred method in these patients since it avoids manipulation of the airway. Neuraxial blockade produces minimal alterations in pulmonary variables in healthy and even in elderly patients. It has no effect on inspiratory function but may produce a decrease in expiratory capacity owing to paralysis of abdominal muscles. [2] Adverse pulmonary complications following neuraxial block are rare.

A few cases of bronchospasm following neuraxial blockade have been reported. [3-6] The causes in these cases have been attributed mainly to high spinal blockade. [3-4] In mid spinal block where bronchospasm has occurred, the causes documented were anxiety, [4] parasympathetic stimulation during surgical manipulation [5] and blockade of sympathetic supply to the adrenal medulla

leading to a decrease in circulating epinephrine levels. [6]

Here we report two cases of bronchospasm occurring under spinal anesthesia in susceptible individuals with absence of high sympathetic block. Possible mechanisms, treatment along with review of literature are discussed.

CASE REPORT

Case 1

A 50 year old male patient was admitted to the surgical ward for cellulitis of the left leg. He had a history of bronchial asthma since 5 years for which he was on levosalbutamol and foracort (formoterol and budesonide) metered dose inhaler 2 puffs twice a day. He had no past surgical history. He gave no history of any allergies and was a nonsmoker. He was taken up for emergency fasciotomy of the left leg after a 6 hour period of oral fasting. Investigations available preoperatively included Complete blood count (Hemoglobin - 14.5 gm/dl, platelet count - 1,88,000/cu.mm, total leucocyte count - 11,000/cu.mm), Renal function test (Blood urea - 126.5, Sr. Creatinine - 2.04), Serum electrolytes (Sodium-134, Potassium-6, Chloride-102), Coagulation Profile (Bleeding time - 2 min, Clotting time - 4 min 40 sec, Prothrombin time - 21.30sec INR -1.78), HIV (non reactive) and HBSAg (non reactive). Chest Xray was suggestive of changes consistent with chronic obstructive pulmonary disease and ECG was unremarkable. Accordingly he was assigned to ASA grade III E.

Inside the operating room, routine monitoring with ECG, Pulse Oximetry and Noninvasive blood pressure was applied. Intravenous access was achieved with 18 G cannula in the left forearm and the patient was started on 0.9% sodium chloride. He was coloaded with 400 ml 0.9% sodium chloride at the time of induction. His vitals prior to administration of subarachnoid block were HR-84/min, BP-124/72 mmHg and SpO2-90% on room air. Chest was clear on auscultation. Rest of the systemic examination was unremarkable. He was administered Inj. Hydrocortisone 100 mg I.V. and Inj. Dexamethasone 8 mg I.V. prior to subarachnoid block in view of history of asthma.

The patient was placed in the left lateral position and a subarachnoid block was achieved with 10 mg of 0.5% hyperbaric Bupivacaine using a 25 G Quincke needle in L3-L4 space under all aseptic precaution. The patient was kept in the left lateral position for 3 minutes to achieve unilateral effect in the left lower limb. The patient was then placed in the supine position and started on 4 liters oxygen via venti mask. The surgery was commenced once adequate effect was achieved.

15 mins after being placed in the supine position he complained of tachypnea, difficulty in breathing and coughing. Oxygen saturation dropped to 77% on 4 litres oxygen via venti mask. On chest auscultation bilateral wheeze was present. He was administered Inj. Hydrocortisone 100 mg I.V. and Inj. Dexamethasone 8 mg I.V. Inj Deriphyllin 220 mg was given slowly I.V. Block height was reassessed at this point & was T8 to pin prick. 5 min later the patient was comfortable, saturation improved to 86%on 6 liter oxygen, however bilateral wheeze although reduced was still present. Patient was then given 2 puffs of Levosalbutamol and Foracort (formoterol and budesonide) via his metered dose inhaler. 8 min later his saturation improved to 92% on 4 liter oxygen and chest was clear to auscultation. The entire episode lasted for about 20 min, after which the surgery was completed successfully. At the time of shifting from the OT he was asymptomatic, hemodynamically stable and SpO2 was 91% on room air.

Case 2

A 27 year old male patient was admitted to the trauma ward with

compound fracture of the right medial malleolus. His past medical and surgical history was unremarkable. He gave no history of any allergies and was a nonsmoker. He was approximately 154 cm in height and weighed 68 kg. After a 6 hour period of oral fasting he was taken up for debridement and fixation of the right medial malleolus on an emergency basis. As he was taken up for surgery as an emergency procedure, as per hospital protocol, investigations available preoperatively were hemoglobin (13.2 gm%), HIV (non reactive) and HBSAg (nonreactive). Accordingly he was assigned to ASA grade I E.

Inside the operating room, routine monitoring with ECG, Pulse Oximetry and Noninvasive blood pressure was applied and his vitals were HR-84/min, BP-124/72mmhg and SpO2-100% on room air. On chest auscultation no foreign sounds were heard. Rest of the systemic examination was also unremarkable. Intravenous access was achieved with 18 G cannula in the left forearm and the patient was started on Ringer Lactate. He was coloaded with 300 ml of Ringer Lactate at the time of induction.

The patient was placed in the right lateral position and a subarachnoid block was achieved with 10 mg of 0.5% heavy Bupivacaine and Tramadol 25mg using a 25 G Quincke needle in L3-L4 space under all aseptic precaution. The patient was kept in the right lateral position for 3 minutes to achieve unilateral effect in the right lower limb. The patient was then placed in the supine position and started on 3 liters oxygen via venti mask.

5 min after being placed in the supine position he began to cough. On chest auscultation bilateral rhonchi were present. His vitals at this time were BP-112/68 mmhg, HR-92/min and SpO2-88% on 6 liters O2. The level of block was found to be T10 by pinprick method. He was administered Inj. Hydrocortisone 100 mg IV and Inj. Dexamethasone 8 mg IV. 3 minutes later his cough and rhonchi persisted and SpO2 decreased to 82% on 6 liter O2. At this point the patient was administered Inj. Deriphyllin 220 mg IV and Inj Pheniramine Maleate 44.5 mg IV. Following this there was only a slight improvement in his condition. A repeat dose of Inj. Hydrocortisone 100 mg IV and Inj Dexamethasone 8 mg IV was given. The patient was then nebulised with Salbutamol and Budesonide. 10 minutes later there was a complete disappearance of his cough and wheeze and SpO2 improved to 98% on 6 liters O2.Surgery was completed and at the time of shifting from the OT he was asymptomatic, hemodynamically stable and SpO2 was 100% on room air. On further questioning the patient gave a history of seasonal rhinitis since the last 10 years which was unrevealed in PAE. He was not on any medication for the same. Chest x-ray done in the immediate post operative period was unremarkable and a differential leucocyte count revealed 0.1% eosinophils.

Both patients underwent an uneventful course postoperatively and were discharged as per hospital protocol.

DISCUSSION

Regional anesthesia is the chosen mode of anesthesia in asthmatic patients wherever possible as it avoids airway manipulation and triggering of bronchospasm.

Allergic rhinitis and bronchial asthma are different manifestations of the same pathophysiological process and need to be treated as a single airway disease. [1] It is proven that patients with allergic rhinitis will have coexisting asthma or develop it at some point in time. Studies have shown the coexistence of allergic rhinitis and bronchial asthma to range from 60 to 100%. [6-8] Patients with allergic rhinitis or bronchial asthma have a generally sensitive upper and lower airway. These patients have been shown to

have autonomic dysfunction with a predominant parasympathetic tone. [9-11]

Occurrence of bronchospasm under spinal anesthesia is rare and most of the time it is associated with high spinal anesthesia. The principal function of sympathetic fibers arising from T1-T4 is bronchodilation. Blockade of these fibers in high spinal anesthesia predisposes the patient for bronchospasm. Mudarradi et al. [3] reported a case of a 20 year old primigravida, known case of asthma who was asymptomatic preoperatively, was taken up for emergency cesarean section under spinal anesthesia who developed bronchospasm intraoperatively. They suggested that asthmatic patients would have an abnormal autonomic nervous system, which is responsible for airway hyperactivity. It is known that the stimulation of the parasympathetic nervous system is implicated in the pathogenesis of asthma. In their case peak sensory level was T4, so expected sympathetic block would be two levels higher i.e. T2. They explained that blockade of this sympathetic supply caused by spinal anesthesia might have triggered the asthmatic attack by influencing the cholinergic ganglia of the lung and decreasing pulmonary blood flow. Similarly, Kawabata et al [4] also reported two cases of bronchospasm in spinal anesthesia in whom peak sensory level was T3, they also attributed the event of bronchospasm to high sympathetic blockade.

In contrast, the peak level of sensory block achieved in our cases was T8 and T10 respectively, so the expected height of sympathetic block would have been 2 levels higher (T6, T8) and high sympathetic block as the cause of bronchospasm as given in above mentioned reports can be ruled out.

Mallampati [6] discussed another mechanism for bronchospasm in mid spinal block height. He reported an occurrence of unexpected bronchospasm in an 18 year old primigravida, a known case of asthma since childhood, who underwent termination of pregnancy in her 12^{th} week under spinal anesthesia. He explained that sympathetic outflow to adrenal medulla arises from spinal nerves T10-L1. A blockade of this sympathetic outflow will lead to a decrease in circulating epinephrine, especially in view of the fact that circulating adrenaline has a very rapid clearance. [12-13] As there is no direct sympathetic innervation to bronchial smooth muscles in humans, [13] the stimulation of beta adrenergic receptors in bronchial smooth muscles is probably provided by circulating catecholamines. Such a decrease in circulating epinephrine will further exaggerate the parasympathetic dominance inherently present in patients with bronchial asthma. This was the possible mechanism in our cases as well.

John B. Pollard [14] in his study on cardiac arrest under spinal anesthesia stated that inadequate preloading can stimulate the vagus via reflex mechanisms (Bainbridge reflex, Bezold Jarisch reflex). In our cases patients were coloaded with only 400ml and 300ml of crystalloid at the time of induction respectively. Although there was no bradycardia in either of our cases, stimulation of the vagus in this way might have contributed to increase in the parasympathetic tone and consequent bronchospasm in these patients where there is already a parasympathetic dominance.

Allergic reaction or anaphylaxis due to a drug could be ruled out as a cause of bronchospasm since at the time of occurrence of bronchospasm there was no significant change in vitals, no flushing of the skin, rash or edema. The patients were not administered any drugs till the occurrence of the bronchospasm. In the immediate post operative period there was no rise in the patients' eosinophil counts.

Prabhakar et al. [5] reported a case of bronchospasm in an otherwise healthy

patient undergoing inguinal hernia repair under subarachnoid block with a maximum block height of T6. Parasympathetic stimulation due to bowel manipulation was speculated as the cause of bronchospasm with anxiety as an additive factor. As the surgeries documented in our case report were lower limb surgeries there were no chances of parasympathetic stimulation as seen in abdominal surgeries.

Both patients in our report were adequately prepared psychologically for the procedure making anxiety [5] as a cause of bronchospasm highly unlikely. Desired effect of spinal anesthesia was achieved and pain as a factor can be ruled out. Also stimulation by endotracheal tube as a factor for bronchospasm was not a possibility in the reported cases.

In our orthopedic case pulmonary embolism from deep veins of the leg was one of the differential diagnoses based on the observed respiratory symptoms. Cases have been reported during orthopedic surgeries under spinal anesthesia in which embolisms were associated with either tourniquet inflation, [15] deflation [16] or surgical manipulation. [17] In our orthopedic case tourniquet was not applied and symptoms occurred prior to surgical manipulation. The patient had complaints of only cough without hemoptysis or chest pain and remained hemodynamically stable throughout. Symptoms were relieved by therapy directed towards bronchospasm without any hemodynamic or pulmonary residual sign and symptoms at the time of shifting from the operating room. Hence pulmonary embolism as a cause of bronchospasm can be excluded.

In all of the above reported cases of bronchospasm [3-6] including ours primary management consisted of the use of bronchodilators (Etophylline + Theophylline, Beta agonist inhalation, Antihistaminics) and steroids. Second line treatment was given for additional contributing factors such as midazolam for anxiety. [4] The role of vagolytics (atropine, glycopyrolate) in the treatment of bronchospasm in patients with a parasympathetic dominance has been discussed by McGough et al in their case report on unexpected bronchospasm during spinal anesthesia. [18] In all the reported cases the patients were successfully treated with the above treatment.

There are no clear guidelines regarding the prophylactic management of patients with bronchial asthma and allergic rhinitis undergoing spinal anesthesia. At our institution patients with a history of bronchial asthma who are to undergo general anesthesia are given prophylactic steroids and bronchodilators even if asymptomatic preoperatively, this practice is not applied in cases of spinal anesthesia. In our first case patient received prophylaxis with steroids which lead to a delayed occurrence of symptoms as compared to our second case where symptoms occurred immediately after injection of drug. This evidence may imply that use of prophylactic bronchodilators and steroids before induction of anesthesia in susceptible individuals may have a protective role to play.

CONCLUSION

In conclusion, bronchospasm should be considered as one of the complications of spinal anesthesia in patients with bronchial asthma and allergic rhinitis. Prophylactic measures should be taken to prevent the occurrence, even if these patients are asymptomatic preoperatively since bronchospasm can be a fatal complication.

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