Ranitidine induced bronchospasm during spinal anaesthesia

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Bronchospasm although is a rare event under spinal anaesthesia, has the potential to become an anaesthetic disaster if not recognized and managed properly. Ranitidine induced bronchospasm is a rare event in anaesthesia practice. We hereby report a case of successful management of bronchospasm under spinal anaesthesia in a 55 year female posted for femoropopliteal bypass grafting because of ranitidine administration.

Key words: ranitidine, bronchospasm, spinal anaesthesia

Introduction

Bronchospasm has the potential to become an anaesthetic disaster if it is not recognized. The incidence of bronchospasm is relatively higher with general anaesthesia, however is a rare event under spinal anaesthesia as there is no airway manipulation. Intraoperative bronchospasm under spinal anaesthesia may occur because of various causes like high level of sensory blockade, drug induced histamine release, parasympathomimetic stimulation (surgical stimulation), prior history of asthma, anaphylaxis or drugs with beta blocking activity. Ranitidine an H$_2$ receptor antagonist has been associated with anaphylactic reactions. We hereby report a case of bronchospasm under spinal anaesthesia in a 55 year female posted for femoropopliteal bypass grafting because of ranitidine administration.

Case report

55 year female having peripheral arterial disease was scheduled for emergency femoro-popliteal bypass grafting because of pain in lower limbs. She was a chronic smoker. There was no previous history of bronchial allergy and any other respiratory complaint. She had no significant medical and surgical history. Blood pressure was 130/80mmHg and pulse was 90/min. Complete haemogram, bleeding time, clotting time, urine examination, blood urea, blood sugar, chest X ray and electrocardiography were normal. Regional anaesthesia (spinal along with epidural) was planned for the procedure. In the operating room, standard monitors were attached. Epidural catheter was placed under aseptic precautions at L$_2$-L$_3$ interspace. Lumbar puncture was done with 23G quincke needle at L$_3$-L$_4$ interspace. Clear CSF (cerebrospinal fluid) was obtained and 2.5ml of 0.5% hyperbaric bupivacaine with 25µg fentanyl was injected. Adequate sensory block was achieved upto T$_{10}$. After 3 hours, patient complained of epigastric pain. Ranitidine 50mg was given intravenously. After 10 minutes, patient complained of shortness of breath. SpO$_2$ was 91%. Auscultation of the chest revealed bilateral rhonchi. Hydrocortisone 100mg was given intravenously and the patient was nebulized with levosalbutamol. Bronchospasm got relieved after 20 minutes. Her SpO$_2$ became 99%. Surgery lasted for 5 hours and postoperative course was uneventful. A provocation test was performed after 3 days with one tablet (150mg) ranitidine. Patient developed diffuse bronchospasm 3 hours after the drug administration which was treated with 100mg hydrocortisone intravenously and nebulisation with levosalbutamol. Patient was advised not to take ranitidine in the future.

Discussion

Ranitidine is commonly used as a preanaesthetic medication in addition to its conventional use in peptic ulcers and hyper secretory states. As compared to other H$_2$ receptor blockers, it has lesser side effects. Development of bronchospasm after intake of ranitidine is an uncommon event. This compound is a substituted aminoalkyl furan without the imidazole nucleus present in other H$_2$ receptor blockers.
Mudaraddi et al reported a case of intraoperative bronchospasm in a patient who was scheduled for emergency caesarean section under spinal anaesthesia due to high level of sensory blockade. The principal function of the sympathetic (T2-T4) supply to the lung is bronchodilation. The stimulation of the parasympathetic nervous system is implicated in the pathogenesis of bronchospasm. A thoracic sympathetic blockade which is made by spinal anaesthesia might trigger bronchospasm by influencing the cholinergic ganglia of the lung and the pulmonary blood flow. This could not be the reason in our case as the level of sensory block was T10. As our patient was having sensory block upto T10, surgical stimulation was also ruled out. Also, patient had no prior history of asthma and was not on beta blockers.

Bronchospasm under spinal anaesthesia can be due to various drugs used. In our patient it might be due to ranitidine used for relief of epigastric pain. In bronchi, H1 and H2 receptors are present and both these play a role in causing bronchospasm. H1 receptors lead to bronchoconstriction and H2 receptors lead to bronchodilatation. H2 receptor blockade prevents the negative feedback of histamine on further mediator release. Ranitidine is an H2 receptor blocker and causes a rise in plasma histamine levels which can lead to bronchospasm. In addition; ranitidine is a substituted aminoalkyl furan. The furan nucleus present in it might be responsible for urticarial lesions also.

To conclude ranitidine induced bronchospasm is a rare event in anaesthesia practice but when it occurs can become an anaesthetic disaster if not recognized and managed promptly.

References
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