An Attack of Bronchial Asthma during Spinal Anesthesia

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• Spinal anesthesia for operations in the lower half of the body is an attractive choice for an asymptomatic patient with a history of asthmatic attacks. It is known that bronchospasm is rarely induced by spinal anesthesia[1]. We experienced an occurrence of attack of bronchial asthma during spinal anesthesia.

Case Report

A 48-year-old woman was scheduled for total hysterectomy with a diagnosis of uterine myoma. She had a history of uneventful appendectomy under spinal anesthesia at the age of 26 year and occasional asthmatic attacks since the age of 26 year. She had been hospitalized three times for treatment of severe attacks of asthma. However, for the past 16 years, she never had severe attack of asthma under administration of salbutamol hemisulfate, clorprenaline HCl, and prednisolone. For recent 2 years, she had only a few episodes of slight difficulty to breath in the morning, which immediately improved by inhalation of isoproterenol sulfate from hand-held delivery units. She had no allergy to drugs. She weighted 55 kg and was 149 cm in tall. Her blood pressure and pulse rate were 134/86 mmHg and 84 beats·min^{-1}, respectively. She had no respiratory symptoms and the chest was clear on percussion and auscultation; she was judged to be in remission of asthma on admission.

No premedication was given. An intravenous catheter was placed after arrival in the operating room, through which 100 mg of hydrocortisone succinate was injected. Spinal anesthesia was uneventfully induced with the patient in the right lateral position using 2.2 ml of hyperbaric solution of 0.3% dibucaine and 0.2 ml of 1:1000 epinephrine injected through a 25-gauge spinal needle at the L2–L3 interspace. The patient was immediately turned to supine position on a horizontal operating table. Three minutes later, the level of anesthesia certified by cold sponge was T4. Blood pressure and pulse rate decreased from 130/90 mmHg to 90/60 mmHg and from 92 beats·min^{-1} to 76 beats·min^{-1}, respectively. Blood pressure and pulse rate immediately recovered after the intravenous administration of atropine sulfate, 0.5 mg, and ephedrine HCl, 10 mg. Fifteen minutes after the spinal anesthesia, the level of anesthesia certified by pin prick extended to T4 bilaterally. She was then placed in the lithotomy position and gynecologists began to operate at approximately 25 min after the start of anesthesia. Shrotly thereafter, she complained of dyspnea. The level of anesthesia to pin prick was immediately retested at this time and was judged to be T4. Auscultation of the chest revealed wheezing bilaterally. Oxygen was administered via face mask. Hydrocortisone succinate, 300 mg, was given intravenously and an intravenous infusion of

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aminophylline was begun at a rate of 250 mg·hr^{-1}. Additional ephedrine HCl, 10 mg, was intravenously administered to stabilize her blood pressure. The asthmatic attack gradually subsided and auscultation of the chest 30 min after the beginning of intravenous aminophylline was clear. It took 150 min to complete the operation. The level of anesthesia certified by pin prick at the end of the operation was T8 bilaterally.

Discussion

In asthmatic patients, the various stimuli to the airway can cause life-threatening bronchospasm. To avoid the possibility of bronchospasm that may be induced by the stimuli to the airway, spinal anesthesia is an attractive choice when the sites of operation are the lower abdominal regions or lower extremities. However, the clinical importance is the fact that unexpected bronchospasm can be induced by high spinal anesthesia.

Several factors may be associated with an occurrence of bronchospasm during spinal anesthesia in asthmatic patients. Airway smooth muscle tone is regulated in part by autonomic nervous system acting on specific receptors: muscarinic and alpha-adrenergic receptors, which are excitatory, and beta-adrenergic and possibly peptidergic receptors, which are inhibitory. In asthmatic patients, autonomic control appears to be abnormal, and the imbalance between excitatory and inhibitory effects may contribute to genesis of asthmatic attacks. The sympathetic block induced by high spinal anesthesia may effect on the control of the autonomic nervous systems. The sensory block was at T4 level when asthmatic attack occurred in this patient. In spinal anesthesia, the sympathetic block exceeds the sensory block two to three segments[2] and is greater when adrenaline is added to the injected solution[3]. The fact that a decrease in heart rate after spinal anesthesia also suggests the blockade of cardiac sympathetic nerves which arise from T1–T4. Therefore, pulmonary sympathetic nerves which arise from T2–T4 may be completely blocked in this case. Parasympathetic (vagal) overactivity may play an important role in the occurrence of asthmatic attack.

There is a inverse relationship between the sensory dermatome level of spinal anesthesia and the changes of plasma catecholamines. High thoracic spinal anesthesia causes suppression of release of norepinephrine and epinephrine from adrenal medulla[4]. The circulating epinephrine act directly on beta-adrenergic receptors in bronchial smooth muscle, or it at indirectly by stimulation of beta-adrenergic receptors on mast cells to inhibit release of mediators such as histamine. The reduced plasma level of epinephrine that dose not cause problems in the absence of bronchial asthma may allow release of histamine and other mediators from sensitized pulmonary mast cells in asthmatic patients[5]. The sympathetic nerves to adrenal gland which arise from T10–L1 were completely blocked at the time of asthmatic attack in this patient. Decrease in plasma level of epinephrine may play a role in the occurrence of bronchospasm.

The psychogenic factors have been implicated in the genesis of bronchospasm. The patient hesitated about whether to receive the operation when her doctor told that total hysterectomy was a conclusive therapy for her uterine myoma. She reluctantly consented to receive the operation, but remained anxious about the operation. However, she was emotionally stable when she arrived in the operating room. Spinal anesthesia was uneventfully performed without any complaint. Psychogenic factors such as anxiousness or excitement were probably not considerable factors in this episode.

Many kinds of drugs and severe pain also have been implicated in the genesis of bronchospasm. No drugs except for hydrocortisone succinate were given to this patient before spinal anesthesia. The patient was free from the discomfort until dyspea occurred. Anesthesia was adequate when the gynecologists began to operate. These factors most probably were not

causative factors in this patient.

We reported an occurrence of attack of bronchial asthma during spinal anesthesia. The clinical importance is the fact that unexpected bronchospasm can be induced by high spinal anesthesia.

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