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Bronchospasm induced with ergometrine during high spinal anesthesia

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Introduction

Ergometrine is useful in the treatment of hemorrhage associated with uterine atony. There are only a few reports of bronchospasm following ergometrine [1,2]. We experienced a case of bronchospasm induced with ergometrine during high spinal anesthesia.

Case report

A 22-year-old woman (gravida 1, para 0) was admitted to our hospital for abortion in her 9th week of pregnancy. Past medical history was unremarkable except for mild asthma since childhood. During adolescence, she had several episodes of breathing difficulty which were improved by inhalation of salbutamol hemisulfate from hand-held delivery units. She had never been hospitalized for treatment of asthma. She had no known allergies to drugs. She was 166 cm tall and weighed 50 kg. Blood pressure was 120/68 mmHg and her pulse rate was 112 beats·min^{-1}. She had no respiratory symptoms and the chest was clear on percussion and auscultation.

No premedication was given. An intravenous catheter was placed to administer lactated Ringer's solution at rate of about 10 ml·kg^{-1}·h^{-1}. Amethocaine 8 mg in 2 ml of 10% glucose solution was injected into the subarachnoid space through a 25-gauge spinal needle inserted at the L3–4 interspace. Five minutes after the spinal injection, the level of analgesia to pinprick extended to T7. She was placed in the lithotomy position. Blood pressure was 110/70 mmHg, her pulse rate was 78 beats·min^{-1}, and percutaneous oxygen saturation (Spo_2) was 100%. Cervical dilation was started 10 min after the spinal injection. She was comfortable during the procedure and the level of analgesia to pinprick was T4. After curettage, ergometrine maleate 0.2 mg was intravenously administered. One minute later, she complained of nausea and shortness of breath, and then became extremely agitated. Blood pressure increased from 100/52 mmHg to 140/90 mmHg and the pulse rate from 82 beats·min^{-1} to 117 beats·min^{-1}. Spo_2 decreased from 100% to 97%. Wheezing was auscultated in the bilateral chest. At this time, the level of analgesia to pinprick extended to T2. There was no eruption or urticaria in the skin. The patient was given oxygen by mask, a bolus injection of aminophylline 125 mg, followed by a rate of about 125 mg·h^{-1}, and intravenous hydrocortisone sodium succinate 500 mg. Bronchospasm gradually subsided and no wheezing in the chest was auscultated 20 min after the onset of the attack.

Discussion

To our knowledge, only two cases of bronchospasm induced with ergometrine have been reported. Sellers and Long [1] reported bronchospasm following ergometrine during general anesthesia in a patient with well-controlled asthma. Crawford [2] also reported bronchospasm following ergometrine during epidural anesthesia in a patient who suffered slightly from asthma in early childhood. Although ergometrine produces contraction of smooth muscle in the organs, it is thought to be of little clinical importance in non-asthmatic patients. However, ergometrine may potentially affect the bronchial smooth muscles in an asthmatic patient.

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Several factors may have been associated with bronchospasm in our patient. Possible factors include pain, psychogenic factors, vagal overactivity, or drug reactions. Pain can be excluded as a causative factor because she was free from surgical pain. Psychogenic factors may have a role in the genesis of bronchospasm, as she was undergoing abortion for an unwanted pregnancy. Although a better sedation might have prevented her fear and anxiety, she appeared to be emotionally stable in the operating theater.

High sympathetic blockade leads to bronchospasm through vagal overactivity [3]. The level of sensory analgesia to pinprick was at T2 when the bronchospasm occurred. The pulmonary sympathetic nerves that arise from T2–T4 were thought to have been completely blocked. Therefore, vagal overactivity induced by high spinal anesthesia might have played an important role in the genesis of bronchospasm in our patient.

A wide variety of drugs also have been implicated in the genesis of bronchospasm. Bronchospasm also occurs in anaphylaxis and anaphylactoid reactions [4]. No drugs were administered to our patient either orally or parenterally before spinal anesthesia. Amethocaine injected into the subarachnoid space produced no allergic reaction. No eruption or urticaria appeared after the administration of ergometrine. Anaphylaxis and anaphylactoid reactions can be excluded as causative factors in this patient.

In a review of 687 patients with a history of bronchospasm [5], the frequency of intraoperative bronchospasm is 3.9%. In patients undergoing regional anesthesia, the frequency of bronchospasm is 1.9%, which is significantly lower than the 6.4% in patients who underwent general anesthesia with tracheal intubation. Indeed, one way to reduce the risk of bronchospasm associated with instrumentation of the airway is use of regional anesthesia. However, regional anesthesia does not guarantee protection from bronchospasm. Several cases of bronchospasm during spinal anesthesia have been reported [6–9].

We think that the bronchospasm in this patient was associated with a combination of administration of ergometrine and vagal overactivity induced by extremely high spinal anesthesia. Anesthesiologists must keep in mind that a combination of these potential factors may produce constriction of bronchial smooth muscle especially in asthmatic patients.

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