PROFOUND PAIN DUE TO PROPOFOL INJECTION TRIGGERED SEVERE BRONCHOSPASM IN A SMOKER

- A Case Report -

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Propofol-induced pain is one of side effects observed during injection[1]. We describe a patient who developed severe bronchospasm triggered by propofol-induced pain during induction of anesthesia.

Case Report

The patient, a 48-years-old smoker woman (10 packs/day), 60kg, without any history of allergy, asthma or cardiac disease, was scheduled for dilatation and curettage for metrorrhagia. Preoperative examination revealed clear lungs, normal heart sounds. Chest radiography and the laboratory findings were normal.

Before induction, the base line heart rate was 78 bpm, blood pressure was 110/85 mmhg and SpO_2 was 100%. Following preoxygenation (6 L/m) and under continuous monitoring, general anesthesia was induced by i.v adminstration of propofol (120 mg over 25 s) through an 18-guage catheter at her forearm. The patient complained of a profound vascular pain during the infusion of propofol associated with withdrawal of her left forearm. During this event, and after loss of consciousness, ventilation through a facemask became impossible; eighty mg of propofol combined with 4% sevoflurane were administered

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immediately. After 3 min, SpO_2 saturation decreased from 100% to 80% and the ventilation remained hopeless. The trachea was immediately intubated, facilitated by succinylcholine 100 mg and fentanyl 100 μg. Chest auscultation revealed bilateral expiratory wheezing with greatly diminished respiratory air entry. Initial peak airway pressure was 50 cm H_2O. She was given six doses of 100 μg salbutamol via the endotracheal tube with aminophyline 100 mg i.v and 8% sevoflurane in O_2. During the ensuing 5 minutes, it became possible to ventilate with small tidal volumes. Oxygen saturation began to increase and wheezing breath sounds were diminished. Additional treatment with hydrocortisone 200 mg i.v and nebulized 500 μg salbutamol was administered via the endotracheal tube. During the next few minutes, manual ventilation became progressively easier with tidal volume increasing to 350-450 ml at pressures of 35-40 cm H_2O. The patient's SpO_2 increased to 100%, breath sound returned to normal and anesthesia was continued with 2%-4% sevoflurane, 3 L/min O_2, 2 L/min N_2O, and no additional muscle relaxants were needed. During this event, her blood pressure ranged between 130/90 and 150/95 mmhg and her pulse ranged between 90 and 130 beats/min. Surgery continued uneventfully. With the patient spontaneously breathing, volumes of 400-500 ml at a respiratory rate of 16 breaths/min, nitrous oxide was discontinued. We elected to extubate the patient after 5 min of 4% sevoflurane in O_2. While 100% O_2 was administered, she continued to breathe spontaneously, became awake, and was transferred to the postanesthesia care unit, where the SpO_2 on room air was 99-100% and her chest was clear. Blood gases and chest radiography were normal. The patient was discharged on the second postoperative day.

Propofol, administered in this case included soybean oil, glycerin, yolk lecithin and disodium edetate.

Propofol is often used in asthmatic patients because of its bronchodilating effects on airway smooth muscle. However, propofol induced bronchospasm during induction of anesthesia have been reported[2,3]. All patients had history of allergic diseases that were well known before induction of anesthesia (allergic rhinitis, atopic dermatitis)

or/and chest disease (sick house syndrome). These patients were later found to have positive allergic test against propofol. In our patient, no history of allergic disease had been noted. Pain, emotional stress, and stimulation during induction of anesthesia can induce bronchospasm. All cases of propofol-induced bronchospasm have been reported without notification if pain was experienced during injection. Propofol formulation has been suggested as a causing factor in this event. The formulations of propofol-containing EDTA or propofol without preservatives (widely available outside USA) have been proposed previously to offer more protection against tracheal intubation-induced bronchoconstriction than the formulation for sulfite-containing propofol. Adverse reactions, including acute asthma and bronchospasm have been reported with several other drugs containing sulfite as preservative.

Furthermore, Rieschke et al[4] demonstrated that tracheal intubation in smokers after induction and maintenance of anesthesia with sulfite-containing propofol produces higher postintubation total respiratory system resistance than when propofol-containing EDTA was used. Our patient, was a known smoker and without history of a previous tracheal intubation. The propofol used on him did not contain sulfite as preservative. The only noted event in this case was the profound pain experienced during propofol administration.

A serious accident associated with propofol-induced pain during induction has been reported recently. Morishima et al[5] reported a myocardial ischemia attack due to profound pain during propofol injection.

In conclusion, our patient demonstrates a case of propofol-induced bronchospasm probably induced by the propofol-induced pain in a smoker and caution should be used in such cases.

References

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