Late-onset pulmonary edema due to propofol

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Pulmonary edema after the administration of propofol has rarely been reported. In this case report, we describe pulmonary edema due to the administration of propofol during a Cesarean section and while in the intensive care unit. The skin tests demonstrated strong positive weal and flare reactions to propofol. The patient was treated successfully with mechanical ventilatory support. This report emphasizes that this fatal complication may be seen with propofol and underlying mechanisms and therapeutic approach are discussed.

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Case report

A 35-year-old, 75 kg, pregnant woman with oligohydramnios and fetal distress was scheduled for an emergency Cesarean section. The patient had no pre-existing medical problems. The laboratory data and physical examination showed no remarkable abnormalities. She had no known drug usage and drug allergy. Standard monitoring (electrocardiogram, pulse oximetry, non-invasive arterial blood pressure) was used. General anesthesia was induced with propofol 2 mg/kg and followed rapidly with rocuronium 0.9 mg/kg. Anesthesia was maintained with oxygen and nitrous oxide in equal portions, and 2% of sevoflurane. Apgar scores stood at 9 and 10 over 1 and 5 min. After the umbilical cord was clamped, 2 μg/kg fentanyl citrate was administered. The operation lasted for 45 min; 15 min before the end of the surgery, mechanical ventilation became difficult and peripheral oxygen saturation was decreased to 94%. Pulse rate was 159 beats/min and blood pressure was 151/100. Arterial blood gases showed pH 7.21, pO2 88 mmHg and pCO2 80 mmHg. Assessment of the anesthetic machine, anesthesia circuit and endotracheal tube revealed no evidence of mechanical obstruction. Auscultation of lungs at the time revealed bilateral moist rales. She had no skin rash or eritema. The patient did not get extubated and was transferred to the intensive care unit. In the intensive care unit, her body temperature was 36.5 °C, pulse rate 158 beats/min and blood pressure 129/87 mmHg. Arterial blood gases showed pH 7.20, pO2 71.8 mmHg and pCO2 51 mmHg; oxygen saturation was 88 despite mechanical ventilation instituted on synchronized intermittent mandatory ventilation mode with FiO2 0.70. A chest radiograph showed diffused bilateral patchy infiltration. A cardiovascular system examination revealed that her heart rate was rhythmic but tachycardic. No murmur was heard. In the second hour, she extubated herself because of agitation, propofol 2 mg/kg and rocuronium 0.7 mg/kg were administered, and orotracheal intubation was done. After intubation, peripheral oxygen saturation was decreased to 80% and severe hypotension 60/37 mmHg occurred; hence inotropic therapy was started. Arterial blood gas was as follows: pH 7.17, pO2 53.5 mmHg and pCO2 1015...
59.2 mmHg. This worsening of the patient’s state after medication made us think about the relationship between medication and pulmonary edema. We performed skin tests for allergy. Skin tests performed for fentanyl and rocuronium were all negative, while propofol demonstrated strong positive weal and flare reactions.

She had complications such as pneumothorax and urinary infection, and her intensive care unit stay was prolonged. On the 27th day, she was discharged from intensive care and chest radiography was normal at discharge.

Discussion

In this case report, we describe pulmonary edema due to the administration of propofol during a Cesarean section and while in the intensive care unit.

The major reason for pulmonary edema to occur during anesthesia was membrane edema, which was due to a leaky alveolar capillary membrane and associated with low filling pressures. Previous reports of anesthesia-induced pulmonary edema have been associated with airway obstruction, gas embolism, cardiac failure, fluid overload, acid aspiration, reactions to blood products and drug hypersensitivity reactions (7, 8). The worsening of the patient’s condition in the intensive care unit after administration of propofol and rocuronium made us consider a relationship between pulmonary edema and these drugs. The skin tests performed for fentanyl and rocuronium were all negative, while propofol demonstrated strong positive weal and flare reactions. We thought that pulmonary edema was due to propofol administration.

The pathogenesis of pulmonary edema associated with propofol remains unclear, while anaphylactoid reaction is the most frequently postulated etiology. Propofol contains a diisopropyl chain and a phenol group, both of which have the potential to cause an allergic reaction. The anaphylactoid reaction may increase vascular permeability and result in acute pulmonary edema.

Acute pulmonary edema has been reported as an adverse effect of intravenous propofol administration in adults. In a case report from Taiwan, a 35-year-old man with nasopharyngeal carcinoma experienced acute pulmonary edema and hypotension after propofol infusion through deep central line insertion (4). Similarly, in a case from Japan, a 61-year-old woman experienced pulmonary edema after electroconvulsive therapy under propofol anesthesia (5). Both of them received continuous intravenous infusion of propofol with no drug overdose. In another case from Taiwan, a 10-month-old boy experienced pulmonary edema before gastrointestinal endoscopic examination (6). The authors explained this event as an anaphylactoid reaction.

Cases of pulmonary edema associated with fentanyl and rocuronium have been described (9, 10). Anaphylaxis is generally an unanticipated severe allergic reaction, often explosive in the onset that can occur during the perioperative period, especially during a surgical procedure when multiple drugs are administered during the conduction of an anesthetic. Cardiovascular symptoms and bronchospasm were the most common clinical features. The clinical manifestations of anaphylaxis are derived from the acute release of mediators from mast cells and basophils (11).

Anaphylactic reactions usually occur seconds to minutes after exposure to the relevant antigen. Late-onset anaphylaxis also occurs. The incidence of prolonged response is unknown, but may be common (12). A case from the United States demonstrated that anaphylaxis might occur 3 days after antigen delivery. Garvey et al. presented that their four cases of chlorhexidine allergy symptoms occurred 20–40 min after induction agents (13). In our case, pulmonary edema forms 30 min after propofol administration during a Cesarean section; also in intensive care after the administration of propofol, peripheral oxygen saturation decreased. These events led us to think of the relief between propofol and pulmonary edema.

Late-onset pulmonary edema due to propofol, an unusual unpredictable adverse reaction, must be kept in mind. Early recognition and proper emergent treatment are essential to reversing this complication.

References


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