ANAPHYLAXIS AFTER PROPOFOL INFUSION FOR PORT-A-CATH INSERTION IN A 35-YEAR OLD MAN

Ming-Han Tsai, Ping-Hung Kuo, Ruey-Long Hong, and Pan-Chyr Yang

Abstract: Anaphylaxis to propofol is rare and has not been previously reported in Asia. We describe a 35-year-old man with nasopharyngeal carcinoma who developed acute respiratory distress and hypotension after propofol infusion for parenteral anesthesia for Port-A-Cath insertion. Chest roentgenogram showed bilateral diffuse alveolar infiltrates. Respiratory failure ensued, and vocal cord swelling was found during endotracheal intubation. Hemodynamic data included a low cardiac index, a low systemic vascular resistance, and a high pulmonary vascular resistance. His condition and the shadows on the chest roentgenogram improved quickly after fluid challenge and the use of vasopressors, antihistamine, and intravenous steroids. Early awareness and appropriate management are necessary to prevent a fatal outcome in patients with propofol anaphylaxis.

Case Report

A 35-year-old man was admitted to the intensive care unit (ICU) because of sudden onset of dyspnea in the recovery room after Port-A-Cath insertion. Nasopharyngeal carcinoma had been diagnosed 2 years earlier and he had received radiotherapy and adjuvant chemotherapy. Metastasis to bone and liver was found in February 1998. A Port-A-Cath was inserted in preparation for chemotherapy on 10 March, 1999. No drug allergy history had been noted. The anesthesia was induced by parenteral propofol 40 mL (400 mg). The course of the operation was smooth, without significant blood loss. However, frothy sputum was expectorated after the procedure. Hypotension (blood pressure 71/50 mm Hg) was found immediately when he was transferred to the recovery room. Intravenous phenylephrine (0.2 mg) was administered, and was repeated 15 minutes later due to persistent hypotension. Betamethasone (Rinderon®) 4 mg was also given. Hemoglobin oxygen saturation (SaO 2) deteriorated to 88% when he was breathing through an O 2 mask with an inspiratory O 2 fraction of 60%. Chest roentgenogram showed bilateral alveolar infiltrates (Fig. 1). He was transferred to the ICU because of refractory hypotension despite an aggressive fluid challenge and administration of vasopressors.

On examination, the patient was acutely ill. Vital signs were as follows: blood pressure 96/47 mm Hg under dopamine 20 μg min⁻¹ kg⁻¹; temperature 38.2°C; pulse 130 and respiratory rate 30/minute. The breath sounds were coarse, without wheezing. There was no skin rash. Arterial blood gas determination revealed a pH of 7.388, PCO2 of 38.4 mm Hg, and PO2 of 70.3 mm Hg on a non-rebreathing mask. The hemogram revealed leukocytosis (white blood cell count 12,620 cells/μL) with left shift (segments 84.8%, eosinophils 2.5%). An endotracheal tube was inserted when swelling of bilateral vocal cords and profuse whitish frothy sputum were found.
The hematocrit was elevated from 31.8 to 36.7% within 1 day. Central venous pressure was 13 cm H\textsubscript{2}O. Hemodynamic data from the Swan-Ganz catheter were as follows: mean pulmonary artery pressure 27 mm H\textsubscript{g}; pulmonary arterial occlusion pressure (PAOP) 11 mm H\textsubscript{g}; cardiac index (CI) 2.62 L\textperiodcentered m\textsuperscript{-2}\textperiodcentered min\textsuperscript{-1} (normal, 2.5–4.0 L\textperiodcentered m\textsuperscript{-2}\textperiodcentered min\textsuperscript{-1}); systemic vascular resistance index (SVRI) 1,832 dyn.s.m\textsuperscript{-2}.cm\textsuperscript{-5} (normal, 1,790–2,390 dyn.s.m\textsuperscript{-2}.cm\textsuperscript{-5}); and pulmonary vascular resistance index (PVRI) 488 dyn.s.m\textsuperscript{-2}.cm\textsuperscript{-5} (normal, 225–315 dyn.s.m\textsuperscript{-2}.cm\textsuperscript{-5}). Bedside cardiac echography revealed good left ventricular contractility, without wall motion or valvular abnormalities. Fluid challenge was continued in the ICU with normal saline and plasmanate (plasma protein fraction), and intravenous betamethasone. Sympathomimetic agents (dopamine and norepinephrine) were also used to maintain hemodynamic stability, and were tapered off smoothly within 1 day. Antibiotics (cefazidime and clindamycin) were administered initially but were soon discontinued because there was no evidence of infection. His fever subsided on the day after admission to the ICU. Follow-up roentgenograms of the chest showed rapid resolution of bilateral infiltrates (Fig. 2). He was extubated on 12 March, 1999, when the blood gas showed pH 7.402, PCO\textsubscript{2} 40.1 mm H\textsubscript{g}, and PO\textsubscript{2} 99.7 mm H\textsubscript{g} (on O\textsubscript{2} cannula 3 L/min). The sputum and blood specimens for culture failed to yield significant growth. He was transferred to the general ward in a stable condition on 14 March and was discharged 3 days later.

After three courses of chemotherapy, the patient underwent a follow-up abdominal computerized tomography scan. However, pulmonary edema occurred again after contrast medium injection. This episode was easily controlled by supportive treatment.

**Discussion**

Propofol contains two moieties with the potential to cause allergic reaction: the diisopropyl side chain and the phenol group [3, 4]. Reaction to propofol is thought to be immunoglobulin E (IgE) dependent [4]. The hemodynamic parameters of our patient were notable for decreased CI and PAOP, with increased PVRI and SVRI. These changes are compatible with anaphylactic syndrome [5, 6], which is characterized by an increase in vascular permeability and is often catastrophic [7].

In this situation, allergy to latex on the surgical gloves should also be considered since the pulmonary edema developed after the procedure for Port-A-Cath insertion. Latex anaphylaxis is also an IgE-mediated reaction, and its diagnosis needs to be confirmed by intradermal skin test, skin prick test for latex, rub test, and latex-specific antibody assay [8]. Nevertheless, this etiology was less likely because the surgical wounds were small and there was little chance for prolonged or frequent contact between the surgical gloves and the subcutaneous tissues during the surgical procedure. Patients at risk of propofol anaphylaxis are those with atopy and with a history of allergic reactions to muscle relaxants or other drugs. Some medical centers use serologic tests for histamine and serotonin and radioimmunoassay for IgE against propofol to diagnose anaphylaxis to anesthetics [4, 9]. The intradermal skin test is the most readily available and generally useful diagnostic test for drug allergy [10]. However, our patient’s parents refused a suggestion that skin test for propofol
be performed. However, judging from the time course and clinical manifestations, propofol was the most likely agent to account for his anaphylactic shock and lung edema.

In conclusion, in patients with unexplained severe hypotension following the use of propofol, anaphylaxis to this agent should always be kept in mind. A past history of allergy may provide important information about the risk of anaphylaxis and a skin test is recommended before using this drug. The optimal treatment includes close hemodynamic monitoring and fluid replacement. The use of epinephrine, glucocorticoids, and antihistamine may be essential in reversing all adverse reactions [11].

References