A case of a life-threatening anaphylactoid reaction during the induction of general anesthesia in a patient scheduled for coronary artery bypass graft (CABG) surgery is reported. Treatment required intravenous epinephrine and the use of a percutaneous intra-aortic balloon pump. Subsequent intradermal testing suggested propofol to be the causative agent.

**CASE REPORT**

A 51-year-old man with a history of unstable angina was scheduled for CABG surgery. He had no history of a prior general anesthetic or allergies. He sustained a Q-wave inferior myocardial infarction 12 years ago, and his ejection fraction was 54%. Current medications were nisoldipine, 5 mg, a calcium channel blocker; celiprolol, 200 mg, a β-blocker; and ranitidine, 150 mg, as well as a continuous intravenous nitroglycerin infusion at a rate of 4 μg/kg/min. One hour before induction, the patient was premedicated with morphine sulfate, 8 mg intramuscularly, glycopyrrolate, 0.2 mg intramuscularly, and ranitidine, 50 mg intravenously. In the operating room, in addition to standard monitors, a 5-lead electrocardiogram (ECG) and a pulse oximeter were placed. A 16-gauge catheter and 20-gauge catheter were placed in a peripheral vein and radial artery under local anesthesia.

Before induction, blood pressure was 140/61 mmHg, and heart rate was 62 beats/min. After preoxygenation, a propofol infusion was started at an initial rate of 0.8 μg/kg/min. Three minutes later, the patient received a bolus of 25 μg of sufentanil and a priming dose of 0.5 mg of pancuronium. The patient was adequately mask ventilated with 100% oxygen. One minute later, blood pressure decreased to 51/26 mmHg associated with tachycardia (heart rate, 119 beats/min) and marked ST-segment depression on the ECG (lead V2). The patient also developed a 1.2% to 3% of the cases. In the third French multicenter study (1992-1994), IgE-dependent anaphylaxis was due to muscle relaxants (39.2%), latex (19%), hypnics (5.9%), benzodiazepines (2.1%), opioids (3.5%), plasma substitutes (5%), antibiotics (1.1%), and other drugs given during anesthesia, such as aprotinin and protamine (2.2%).

Six weeks later, in the absence of any drug altering skin reactivity and under full hemodynamic monitoring, all the anesthetic drugs used during the induction of anesthesia were tested by intradermal skin testing as described by Fisher. The incidence of anaphylactic reactions occurring during general anesthesia has been estimated to be 1 in 6,000, propofol being responsible for about 1.2% to 3% of the cases. In the third French multicenter study (1992-1994), IgE-dependent anaphylaxis was due to muscle relaxants (39.2%), latex (19%), hypnics (5.9%), benzodiazepines (2.1%), opioids (3.5%), plasma substitutes (5%), antibiotics (1.1%), and other drugs given during anesthesia, such as aprotinin and protamine (2.2%).

Intradermal testing with propofol at a concentration of 100 μg/mL (10-3) was consistently negative. At the concentration of 1 μg/mL (10-6), a positive test result can be interpreted as a local IgE-dependent histamine release.

Several studies have also demonstrated more serious and frequent anaphylactic reactions in patients receiving β-blockers. The production of anaphylactoid mediators is modulated by neurohumoral mechanisms acting by cyclic nucleotides. β-blockers interfere with these systems so as to amplify the signal and the response. Treatment of anaphylaxis in patients exposed to β-blockers may be difficult and refractory. β-blockade act to block the expected actions of epinephrine, facilitating unopposed α-adrenergic and reflex vagotonic effects that can lead to increased release of mediators. The unopposed α-receptor activation in the presence of excess epinephrine may constrict coronary arteries or exaggerate systemic pressor effects of epinephrine. In the present case, epinephrine had to be used along with an intra-aortic balloon pump for hemodynamic stabilization because the patient, with a history of unstable angina, also had ST-segment depression.
REFERENCES


