CASE REPORT

Case of Vasovagal Syncope With Asystole Associated With Propofol Sedation

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Few cases of bradycardic complications occurring under intravenous sedation have been reported. Here, we report a case of vasovagal syncope with asystole (7.2 seconds) associated with propofol sedation.

Key Words: Vasovagal syncope; Propofol sedation.

The vasovagal response can be triggered by stress, prolonged standing, extreme emotions, or severe pain. It is caused by reduced arterial pressure and blood supply to the brain and is mediated through neural mechanisms rather than primary cardiac dysfunction. Most modern anesthetic agents do not have anticholinergic or sympathomimetic side effects. Simple vasovagal reflexes with bradycardia and transient asystole are more common. Bradycardic complications have been reported to occur after induction, during, or at the end of propofol-induced anesthesia. Abrupt, unpredictable, or progressive decreases in heart rate, as well as cases of sudden cardiac arrest, under general anesthesia have been reported. However, few cases of bradycardic complications occurring under intravenous sedation have been reported. Here, we report a case of vasovagal syncope with asystole (7.2 seconds) associated with propofol sedation.

CASE REPORT

The patient was a 36-year-old woman, 41 kg in weight and 153 cm in height. Since adolescence she had experienced phobia and excessive gag reflex during dental treatment. She had undergone dental treatment under inhalation sedation using nitrous oxide at a nearby dental office in the past. She came to our hospital because her gag reflex had progressed significantly and the nearby dental office no longer used inhalation sedation using nitrous oxide. To treat several carious teeth, management including inhalation sedation was scheduled at our hospital.

She underwent dental treatments under inhalation sedation twice at our hospital. Inhalation sedation was maintained using 30% nitrous oxide in oxygen. She experienced severe postoperative nausea both times, so we planned intravenous sedation for subsequent dental extractions. Intravenous sedation was accomplished with midazolam and propofol, with no complications perioperatively. We also applied intravenous sedation during the next dental treatment, because she stated that she felt more comfortable during the dental extraction with intravenous sedation compared with inhalation sedation.

She was therefore scheduled to undergo her final dental treatment. Propofol sedation was planned to ensure rapid emergence from sedation. She was lying on a dental chair in a supine position. Routine monitoring, including blood pressure (BP), arterial oxygen saturation (SpO2), and an electrocardiogram (ECG), was conducted. Preoperative vital signs revealed a heart rate of 72 beats/min (bpm), BP of 126/74 mm Hg, and SpO2 of 100% (Figure 1). The first attempt at venipuncture was not successful. Immediately after the second venipuncture, the patient’s heart rate was 68 bpm, BP was 124/84 mm Hg, and SpO2 was 100%. We started propofol sedation (target concentration in blood 2.0 µg/mL) using...
a target-controlled infusion (TCI) system, because her vital signs were still stable. About 1 minute later, she indicated that she felt better and comfortable (target concentration in blood 0.4 μg/mL). However, immediately after this confirmation, her HR and BP dropped to 38 bpm and 84/44 mm Hg, respectively, and she lost consciousness. Her ECG (Figure 2) showed transient asystole (duration 7.2 seconds). We first checked the ECG cable and electrodes to ensure they were working properly and then initiated chest compressions. Spontaneous ECG readings (63 bpm) occurred, and her BP and SpO2 still could not be measured at that time. Thereafter, we stopped propofol infusion and administered 0.5 mg of atropine sulfate intravenously. We checked for her left radial pulses while the atropine sulfate was administered, and we could not palpate radial pulses.

Two minutes after the atropine injection, her HR and BP were 87 bpm and 103/58 mm Hg, respectively (Figure 3). We applied midazolam sedation after her vital signs and consciousness had returned to normal and became stable. She showed no abnormal findings thereafter.

After she recovered consciousness, we rechecked her past history. She had fainted during venipuncture during puberty.

**DISCUSSION**

Vasovagal syncope is the loss of consciousness caused by reduced arterial pressure and blood supply to the brain and is mediated through neural mechanisms rather than primary cardiac dysfunction. Bradycardia and vasodilation are the characteristic changes that cause systemic hypotension. The trigger may be central, from psychological stress or pain, or it may be initiated peripherally by a reduction in venous return to the heart.5 The vasovagal response can be triggered by stress, prolonged standing, extreme emotions, or severe pain in patients who are predisposed to venous pooling.6 Prolonged standing7 or orthostasis3 may trigger the vasovagal response as a result of cerebral hypoperfusion.

Our patient developed bradycardia with asystole (7.2 seconds) after TCI propofol administration. True vasovagal reactions have been reported to occur around the time of induction3,8 and during maintenance8 of general anesthesia; however, few cases of reactions occurring under intravenous sedation have been reported. Oei-Lim et al9 reported that bradycardia was not observed during upper gastrointestinal endoscopy using propofol sedation (3.5–4.5 mg/kg/hr = 58–75 μg/kg/min). On the other hand, Souron et al10 reported a 5.7% incidence of hypotensive/bradycardic events during shoulder surgery in the sitting position. Propofol has been reported to produce a 2.0% incidence of bradycardia at induction and an additional 2.8% incidence during anesthesia maintenance.10 Abrupt, unpredictable, or progressive decreases in heart rate and cases of sudden cardiac arrest have been reported.8 Bradycardic complications may occur after induction of, during, or at the end of propofol-induced anesthesia.

There is a possibility that propofol promoted vasovagal syncope. Yorozu et al4 reported that although bradycardia itself rarely progresses to cardiac arrest, propofol reduces heart rate and occasionally induces bradycardia. Propofol can increase the risk of bradycardia, asystole, and death from bradycardic events, because it significantly increases the risk of bradycardia compared with other anesthetics.8 There is an inverse relationship between plasma propofol concentration and cardiac output during continuous infusion. An increase in plasma propofol concentration results in a decrease in cardiac output.11 Win et al5 reported that a decrease in heart rate during propofol sedation is probably attributable to the dominance of parasympathetic action. Transient asystole was observed in our patient. This asystole with bradycardia may have been a result of the
intense effect of parasympathetic nerve tone on the cardiovascular system.

On the other hand, in many cases, the vasovagal response occurs immediately during or after venipuncture, and patients typically complain of nausea, discomfort, or dizziness. Pain and anxiety at venipuncture might trigger an intense parasympathetic state leading to bradycardia and hypotension. However, in the present case, the patient expressed comfort after propofol administration. Therefore, this patient’s clinical presentation could not be explained completely by the vasovagal response from venipuncture.

CONCLUSION

We encountered a case of vasovagal syncope with asystole during sedation with propofol. There is a possibility of bradycardia occurring with asystole, not only during general anesthesia with propofol, as has been reported, but also during intravenous sedation using propofol.

REFERENCES