A Case of Cardiac Arrest for 31 Seconds During Recovery After Intravenous Sedation

Kaoruko Hamazaki, DDS,* Yasuhiko Kato, DDS, PhD,† Akari Hasegawa, DDS,* Hiroko Yoneda, DDS,* Nahoka Miyatani, DDS,* and Yoshihiro Momota, DDS, PhD‡

*Department of Anesthesiology, Osaka Dental University, Osaka, Japan, †Associate Professor, Department of Anesthesiology, Osaka Dental University, Osaka, Japan, and ‡Professor, Department of Anesthesiology, Osaka Dental University, Osaka, Japan

A 26-year-old woman with a history of feeling nauseated during dental local anesthesia presented to our clinic for tooth extraction under intravenous sedation. Although she had experienced episodes of neurally-mediated syncope, her symptoms were controlled well with drug therapy, stopped 3 years earlier. No syncope episodes developed over the previous 2 years. Tooth extraction was performed under intravenous sedation without incident. When she was returned to a sitting position after being roused, convulsion, loss of consciousness, and cardiac arrest developed. One week later, similar symptoms occurred immediately after suture removal. We suspect that the change in body position triggered these episodes. It is important to avoid abrupt changes in body position and any other triggers and to administer preventive drugs in patients at high risk of syncope.

Key Words: cardiac arrest, neurally-mediated syncope, intravenous sedation

Neurally-mediated syncope (NMS) may be triggered by mental or physical stress and may lead to severe bradycardia. We report a case of NMS leading to cardiac arrest lasting for 31 seconds in a patient with dental phobia who underwent intravenous sedation during her dental treatment.

CASE REPORT

A 26-year-old woman (height 150 cm, weight 48 kg) was referred to our oral surgery clinic for the removal of impacted right upper and lower wisdom teeth. As she had felt nauseated during dental treatment with local anesthesia in the past, we considered her event as psychogenic in nature and planned to perform tooth extraction under intravenous sedation.

Her medical history revealed that she had fainted from a young age when she received vaccines or blood sampling or even when she saw others receiving injections. She was diagnosed with NMS at an early teen age and had continued drug therapy until the age of

23 years but could not remember the name of the medication. Her last syncope before visiting our clinic developed at the age of 24 years. She had no family history of syncope and was otherwise healthy.

On the day of the procedure, standard monitors, including electrocardiogram, noninvasive blood pressure, and pulse oximeter were placed. At baseline, her electrocardiogram showed sinus rhythm, blood pressure was 120/65 mm Hg, heart rate was 72 bpm, and oxygen saturation was 98% on room air. Intravenous access was established via a peripheral vein in the dorsum of the hand, and midazolam 3 mg was administered. Her sedation level was 3 on the Ramsay score. Local infiltration anesthesia was initially performed with 3.6 mL 2% lidocaine with epinephrine 1:80,000. No significant changes in vital signs were observed during the procedure. The impacted teeth were extracted using a total volume of 6.8 mL of the lidocaine-epinephrine solution. The procedure was completed in 35 minutes. After tooth extraction, her hemodynamic parameters were confirmed to be normal. However, delay in recovery from sedation was observed. Flumazenil 0.5 mg was therefore administered. About 10 minutes after flumazenil administration, she could communicate appropriately. Her position was changed from the supine to sitting position, and a Romberg test (one criteria for removing monitors in our clinic) was performed. When she was placed in the sitting position after confirming the absence of dizziness, she com-

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Address correspondence to Dr Kaoruko Hamazaki, 1-5-17 Otemae, Chuo-ku, Osaka, 540-0008, Japan; Kaoru.za.0311@gmail.com.

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Figure 1. The electrocardiogram monitor after sedation.

plained of feeling nauseated. She had an abrupt decrease in heart rate, slurred speech, tremor of the extremities, oculogyric crisis, and lost consciousness. The electrocardiogram monitor showed cardiac arrest (the Figure). Immediately after the onset, we placed her in the supine position and started to give chest compression. About 30 seconds after the onset, her heart restarted, and she responded to verbal stimuli. After she became conscious again, she was monitored for about 15 minutes in the supine position. After vital parameters and mental status were confirmed to be stable, we established that her parents were able to accompany her home, and she was discharged. A cardiology referral was made.

One week later, she visited our clinic again for suture removal. With her previous episode of lost consciousness, monitors were placed before removing the sutures. An external cardioverter-defibrillator was kept ready for emergency use. Before the procedure, her blood pressure was 125/65 mm Hg, heart rate was 75 bpm, and oxygen

saturation was 99%. When her position was changed from the supine to sitting position and monitors were being removed after suture removal, she suddenly complained of feeling nauseated. An abrupt decrease in heart rate, clonic spasms of the extremities, and loss of consciousness developed again. She was immediately placed in the supine position and airway, breathing, pulse, and electrocardiogram were evaluated, revealing loss of pulse, cardiac electrical activity, and respiration. As transcutaneous pacing pads were being prepared, pulse and electrocardiogram activity resumed spontaneously, and she regained consciousness. Chest compressions were not initiated because of our experience in the first case, which led us to expect that cardiac activity would spontaneously restart. After she regained consciousness, she was monitored for about 20 minutes in the supine position. After vital parameters and mental status were confirmed to be stable, she was allowed to go home.

The cardiologist diagnosed her with neurally mediated cardioinhibitory syncope but did not prescribe a pacemaker or regular drug therapy. He recommended atropine pretreatment in the future.

DISCUSSION

NMS is the most common cause of idiopathic transient loss of consciousness in individuals without cardiovascular or overt central nervous system disorder. It includes vasovagal syncope (VVS), carotid sinus syndrome, situational syncope that develops in specific situations (such as urination, coughing, swallowing, eating, and blood sampling), and emotional syncope that is induced by emotional stress such as fear, pain, and fright.^{1–3} VVS is classified as a cardioinhibitory form that results in transient bradycardia leading to syncope, a vasoinhibitory form that results in transient hypotension (without bradycardia) leading to syncope, and a mixed form that results in both bradycardia and hypotension.¹

The head-up tilt test has been used in the diagnosis of VVS.^{4,5} This test assesses whether syncope occurs when the patient at rest in the supine position on the table is tilted at different angles.^{4,5} VVS is managed with patient education on lifestyle and measures to avoid syncope, drug treatment using fludrocortisone, alpha-agonists, beta-blockers, disopyramide, and serotonin-reuptake inhibitors as well as nonpharmacological treatment such as an implantable pacemaker and tilt training.^{6–10} The long-term outcome of patients with VVS is relatively good, and no deaths have been reported in a trial with a mean follow-up of 30 months.¹² Although VVS generally does not cause death directly, it may be responsible for injuries and traffic accidents. Implantable pacemakers are considered to prevent cardiac arrests in patients in whom VVS results in long-lasting cardiac arrests and convulsions.⁶

In the present case, the patient had dental and medical phobia with a history of feeling nauseated during dental treatment under local anesthesia. Hence, she underwent tooth extraction under intravenous sedation in our clinic, which was uneventful intraoperatively. It has been reported that a patient experienced episodes of vasovagal response, resulting in sinus arrest due to an insufficient level of sedation during dental treatments under intravenous sedation.¹³ However, we believe that our patient was sufficiently sedated at a Ramsay score of 2 to 3 during tooth extraction preceding the development of the first episode of NMS. The first episode of syncope during recovery was considered unlikely to be directly related to flumazenil treatment, as it developed considerably after drug administration and with good

evidence of sedation reversal. Administration of flumazenil may cause a rise in blood pressure, headache, excitement, or nausea. For more worried and anxious patients, administration of flumazenil may lead to sedation, panic, or an anxious condition. However, only after the Romberg test with changes in body position did syncope develop. The acute arousal with flumazenil, hospital setting, and intravenous access might have caused mental stress, however, and contributed to NMS.

However, the second episode developed with no significant dental interventions. She lost consciousness while talking normally after suture removal. Her syncopal episode occurred when her position was changed from the supine to sitting position, which resembles that during the head-up tilt test. Although our patient appeared to have positionally-induced syncope, stressors such as pain, nervous tension, and anxiety associated with even minor dental treatment may have induced VVS. She did not report syncope in nonmedical situations.

In general, NMS is treated with drug therapy, tilt training, or pacemaker therapy if cardiac arrest is prolonged.⁶ As this patient was relatively young and known to have syncope only in hospitals or clinics, regular drug therapy or pacemaker therapy were not considered in this case. We may be able to prevent syncope episodes in the future by allowing her to become comfortable with the hospital environment to reduce her mental stress and by administering parasympatholytic drugs as premedication. Given our experience in the first case, we might have avoided the second case by administering atropine or light sedation, but we felt this was not necessary for suture removal. Very slow change from the supine to sitting positions might also have avoided her syncopal episode.

In summary, we experienced a case of Neurally-mediated Syncope leading to cardiac arrest lasting for more than 30 seconds in a patient after intravenous moderate sedation. Patients with a history of NMS should be treated carefully to reduce their mental stress in the hospital setting, and an abrupt change in body position should be avoided.

REFERENCES

1. Grubb BP, Karas B. Clinical disorders of the autonomic nervous system associated with orthostatic intolerance: an overview of classification, clinical evaluation, and management. *Pacing Clin Electrophysiol*. 1999;22:798–810.
2. Benditt DG. Neurally mediated syncopal syndromes: pathophysiological and clinical evaluation. *Pacing Clin Electrophysiol*. 1997;20:572–584.
3. Task Force for the Diagnosis and Management of Syncope, European Society of Cardiology (ESC), European

Heart Rhythm Association (EHRA), et al. Guidelines for the diagnosis and management of syncope (version 2009). *Eur Heart J*. 2009;30:2631–2671.

4. Almquist A, Goldenberg IF, Milstein S, et al. Provocation of bradycardia and hypotension by isoproterenol and upright posture in patients with unexplained syncope. *N Engl J Med*. 1989;320:346–351.

5. Inoue H, Aizawa Y, et al: Guidelines for diagnosis and treatment of syncope (JCS 2007) [in Japanese]. *Circ J*. 2007; 71(suppl IV):1049–1101.

6. Mizumaki K. Neuroregulatory syncope: vasovagal reflex. *J Showa Med Assoc*. 2011;71:530–540.

7. Sra JS, Jazayeri MR, Avitall B, et al. Comparison of cardiac pacing with drug therapy in the treatment of neurocardiogenic (vasovagal) syncope with bradycardia or asystole. *N Engl J Med*. 1993;28:1085–1090.

8. Sheldon R. Role of pacing in the treatment of vasovagal syncope. *Am J Cardiol*. 1999;84:26–32.

9. Connolly SJ, Sheldon R, Roberts RS, et al. The North American vasovagal pacemaker study(VPS): randomized trial of permanent cardiac pacing for the prevention of vasovagal syncope. *J Am Coll Cardiol*. 1999;33:16–20.

10. Cox MM, Perlman BA, Mayor MR, et al. Acute and long-term beta-adrenergic blockade for patients with neurocardiogenic syncope. *J Am Coll Cardiol*. 1995;26:1293–1298.

11. Strasberg B, Rechavia E, Sagie A, et al. The head-up tilt table test in patients with syncope of unknown origin. *Am Heart J*. 1989;118:923–927.

12. Baron-Esquivias G, Errazquin F, Pedrote A, et al. Long-term outcome of patients with vasovagal syncope. *Am Heart J*. 2004;147:883–889.

13. Daigo E, Sakuma Y, Kaneda K, Hirokane M, et al. Multiple episodes of vasovagal syncope during dental treatments with intravenous conscious sedation. *J Japan Dent Soc Anesthesiol*. 2010;38:295–300.