Transient asystole associated with vasovagal reflex in an oral surgery patient: A case report

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Abstract

The perioperative cardiac events may be brought about by a relative imbalance of autonomic activities due to excessive psychological and physical stress. The present case study focuses on the asystole that can occur as a serious cardiac adverse event associated with vasovagal reflex likely to be triggered by venipuncture for securing an intravenous line during dental care. In addition, we describe and discuss herein the management of intravenous sedation for a dental phobic patient who experienced the vasovagal reflex involved in an unexpected transient asystole. The patient with vasovagal reflex episodes in daily life, who had no past medical history relevant to cardiovascular disorders, was scheduled for dental extraction under intravenous sedation. Immediately after peripheral intravenous catheterization, she complained of discomfort and nausea, and a II-lead electrocardiogram revealed asystole following bradycardia associated with vasovagal reflex. Oxygenation and intravenous fluid loading in the supine position with elevation of the lower extremities restored sinus rhythm and normal hemodynamics without the intervention of cardiopulmonary resuscitation. With administration of intravenous atropine and betamethasone as premedication, she was uneventfully treated in stress-free psychosomatic conditions under optimal sedation with midazolam without any signs of cardiovascular disorders. After administration of flumazenil, the patient satisfactorily recovered from sedation without re-sedation. The present case suggests that an asystole associated with vasovagal reflex can be triggered by venipuncture for intravenous catheterization during dental anxiety likely to affect the imbalance between sympathetic and parasympathetic activities.

Keywords

Asystole associated with vasovagal reflex, painful peripheral intravenous catheterization, dental anxiety, intravenous sedation, psychosomatic stress-free dental care

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Introduction

The perioperative serious electrocardiographic changes may be originated by a relative imbalance of autonomic activities due to excessive psychosomatic stress during dental care, or anesthetic and surgical procedures.^{1–3} This study case shows a rare and unusual situation of the transient asystole as a serious sign of vasovagal reflex (VVR) likely to be triggered by venipuncture for peripheral intravenous access placement during anticipatory dental anxiety. On the best of our knowledge, there is no report regarding the management of intravenous sedation (IVS) during dental care in a dental phobic patient with the occurrence of asystole. We focus herein on the asystole that can occur as a cardiac adverse event associated with VVR and also discuss the importance of perioperative stress-free management including the appropriate procedure of intravenous catheterization in dental phobic patients particularly with VVR episodes.

Case presentation

A 38-year-old dental phobic woman without needle phobia was scheduled for the extraction of left mandibular third molar under IVS. According to a close medical interview,

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Figure I. (a) II-lead ECG findings revealing the change from sinus bradycardia to transient asystole associated with VVR followed by recovery to sinus rhythm via AVJR. (b) II-lead ECG during IVS management after recovering to normal sinus rhythm (blood pressure: 109/63 mmHg, heart rate: 83 bpm). The ECGs in (a) and (b) were taken by the same II-lead device. Oxygenation and intravenous fluid loading in the supine position with elevation of the lower extremities successfully restored sinus rhythm and normal

hemodynamics without the intervention of cardiopulmonary resuscitation including administration of intravenous atropine and adrenaline, chest compressions, and defibrillation.

she had some fainting episodes in daily life but had never undergone the specific examinations such as central nervous system tests to diagnose a cause of her fainting, which was restored in a few seconds by relaxation in the supine position. Immediately after the occurrence of fainting, her pulse was not checked by anyone, and no one could confirm that fainting episodes were not associated with asystole. However, she had no past or family medical history responsible for asystole relevant to cardiovascular diseases causing VVR and had no problems in regular physical examinations including the consultation of physician. Based on such systemic backgrounds, the patient was judged to have a negative family anamnesis for sudden cardiac death. Therefore, we evaluated that her fainting episodes within a few seconds were non-cardiogenic and likely to be caused by severe bradycardia due to VVR with less possibility of asystole. From this perspective, we did not suggest a cardiologic visit to the patient before scheduling the dental extraction under IVS. Although, in case that the patient suffers from more frequently repeated VVR in her daily life, we think that a preoperative consultation with a cardiologist or neurologist, and a thorough examination for syncope should have been conducted. Besides, more detailed and specific medical examinations including the assessment of a possible orthostatic hypotension related to vasovagal syncope such as the head-up tilt table test,⁴ which is useful and worthwhile for diagnosis of autonomic nerve function, should be required in the future. Preoperative blood biochemical examination revealed no abnormalities, and electrocardiogram (ECG) indicated normal sinus rhythm (69 beats per minute; bpm).

An intravenous line was secured as painlessly as possible during catheter insertion after 30 min of lidocaine (18 mg) tape application under monitoring of non-invasive blood pressure (118/76 mmHg) and II-lead ECG (normal sinus rhythm, heart rate:73 bpm). The patient showed no clinical signs of hypovolemia or dehydration suggesting an indication for administration of Ringer's solution before the emergency but we administered lactated Ringer's solution as an appropriate intravenous fluid to ameliorate potential hypovolemia resulting from same-day fasting prior to IVS.

Immediately afterwards, she complained of some discomfort and nausea, and then the ECG showed the change from serious bradycardia below 30 bpm to transient asystole for 18 s associated with VVR (Figure 1(a)), followed by a loss of consciousness for several seconds. We observed these clinical signs under a preparation of cardiopulmonary resuscitation including administration of intravenous atropine and adrenaline, tracheal intubation, chest compressions, and defibrillation. After a few seconds of convulsion, she responded to our voice and regained consciousness and spontaneous breathing. Prompt oxygenation, intravenous fluid loading by lactated Ringer's solution, and relaxation in

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flumazenil(mg)										0.2+0.3							
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Figure 2. Perioperative management record.

The patient experienced a transient asystole associated with VVR immediately after peripheral intravenous catheterization for IVS. After restoration of normal sinus rhythm, the patient was uneventfully treated with optimal sedation under premedication of atropine and betamethasone with no signs of cardiovascular and respiratory disorders.

the supine position with elevation of the lower extremities without the intervention of cardiopulmonary resuscitation successfully restored sinus rhythm via atrioventricular junctional rhythm (AVJR) (Figure 1(a)) (blood pressure: 111/67 mmHg, heart rate: 75 bpm). Considering that the patient had no problems in systemic conditions as well as in medical history and periodic physical examination, we assessed that the asystole was not cardiogenic but due to VVR based on high parasympathetic hyperactivity and then decided to perform the scheduled dental surgery under IVS with careful systemic management. On the contrary, we judged that the friendly and aforementioned painless approach to the patient could create a state of relaxation without premedication of an oral anxiolytic. With the

preparation of a supraglottic airway device (laryngeal mask), tracheal tube, and defibrillator, atropine 0.25 mg as a parasympathetic blocker and betamethasone 4 mg as an antishock agent were administered for premedication. Sedation was induced and maintained with administration of midazolam (total dose: 2 mg) in the oral surgery ambulatory care unit, under the auspices of the first author, a certified dental anesthesiology specialist. The patient was uneventfully treated with optimal sedative condition of Ramsay sedation score: 3⁵ with no signs of cardiovascular and respiratory disorders. Her blood pressure (90–110/58–75 mmHg), heart rate (65– 85 bpm), electrocardiographic finding (normal sinus rhythm), and oxygen saturation (99%–100%) were stable throughout the stress-free management of IVS (Figure 1(b)). Recovery from sedation after administration of flumazenil 0.5 mg was satisfactory without re-sedation (Figure 2).

Discussion

The patients undergoing dental surgery often experience intense anxiety or phobia, imaging the previous aversive memories of painful stimulation.^{6,7} Related to this point of view, the occurrence of systemic complications comprising hyperventilation syndrome, aberrant hypertension, tachyarrhythmias, or cardiac events such as asystole associated with VVR should be usually taken into much consideration during stressful dental care. VVR characterized by the symptoms of facial pallor, cold sweat, dimmed vision, nausea, bradycardia, and hypotension, which can be induced and exacerbated by a relative imbalance of autonomic activities, is one of the most frequent systemic accidental events in close relationship with injections or painful dental manipulations. In the present patient whose pain threshold may be psychoneurologically lowered because of dental anxiety,⁸ we infer that parasympathetic hypertonia triggered by pain stimulus due to peripheral intravenous catheterization under psychologically stressful dental practice environment played an important role in asystole progressing to vasovagal syncopal event. This pathophysiological mechanism is considered to be responsible for the Bezold-Jarisch reflex that gives rise to hypotension, bradycardia, or cardiac arrest due to VVR.9 Such vasovagal reaction is classified into cardioinhibitory type leading to syncope with transient bradycardia, vasodepressor type leading to syncope only by transient hypotension without bradycardia, and mixed type with both bradycardia and hypotension. Therefore, in the present psychosomatic stress-free management during IVS, we recognize that sinus rhythm was maintained because an adequate balance of autonomic nerve activities with neither sympathetic nor parasympathetic predominance was fulfilled by the optimal sedative effect that can result in higher pain threshold than before IVS.

IVS as a stress-free dental management is esteemed to be a clinically efficacious strategy for providing dental phobic patients with both emotional and hemodynamic stabilization. In this regard, oral administration of a sedative such as diazepam for premedication, or not only analgesia using surface anesthesia but nitrous oxide inhalation sedation to prevent VVR when securing the intravenous line should have been also implemented for a possible relief of the present patient's psychological and painful stress. We reacknowledged that patients with a history of syncope require such measures and much more careful systemic evaluation before treatment in order to avoid very serious complications. Furthermore, it is indicated that the utilization of dental anxiety score assessment, the considerate approach to patients, and audiovisual devices to create a state of relaxation can be recognized as a patient-first perioperative care to overcome dental phobia.^{10,11} With respect to IVS-related medicines, in patients with potentiality of VVR such as the present case, we recommend that the use of propofol as a sedative can be avoided because of its higher predominance of sympathetic hypotonia leading to the incidence of bradycardia and asystole.

We need to distinguish and diagnose the electrocardiographic changes that suggest the possible signs associated with VVR such as AVJR¹² or asystole resulting from parasympathetic hypertonia owing to painful stress of venipuncture for peripheral intravenous catheterization or local anesthetic injection to oral mucosa. On the contrary, it is also reported that the cerebral hypoperfusion causes unconsciousness approximately 6 to 8 s after asystole.¹³ In this viewpoint, the duration of a loss of consciousness owing to asystole can be estimated to be approximately 10 s, judging from the time series of the ECG changes shown in Figure 1(a). Such duration seems to be almost coincident with an image of the actual clinical course.

The sinus node is mainly controlled by the right vagus nerve, while the atrioventricular node is controlled by the left vagus nerve. Hence, a vagal hypertonia, which provokes bradycardia and hypotension due to vasodilation accompanied by a loss of consciousness,¹⁴ can be a major latent factor as an etiology of asystole derived from some clinical situations of surgical and/or general anesthetic stress.^{3,15} In the light of the report that adequate oxygenation can prevent the bradycardia in response to tracheal suction,¹⁶ the administration of oxygen before an exposure to invasive stimulus might be effective for the prophylaxis of bradycardiac arrhythmias. The cardiac adverse events associated with hypotension, bradycardia, and asystole owing to vagal hypertonia require oxygenation and intravenous fluid loading in the supine position with elevation of the lower extremities and medication with atropine as a parasympathetic blocker, etilefrine or ephedrine as a vasopressor, steroids for the prevention of neurogenic shock, or transcutaneous external pacing.¹⁷ It is also essential to administer adrenaline early in established cardiac arrest. In addition, taking into account the report that the ventricular fibrillation following prolonged asystole needed defibrillation,¹⁸ it may be important to bear in mind a possible occurrence of ventricular fibrillation secondary to vasovagal syncope under a preparation of defibrillator. Moreover, a slight ventricular fibrillation masked by an asystole-like waveform may be latent on the ECG, so it is also useful to increase the sensitivity of the ECG waveform to make a differential diagnosis between the two. Pondering whether asystole is cardiogenic or due to other factors such as cerebral disorders, a clinical differential diagnosis of the cause of asystole is indispensable for an adequate therapy. The preoperative general conditions such as hypovolemia due to dehydration, or anemia, which are involved in the systemic risk factors triggering cardiac events, should be recognized and improved before surgery.³ Therefore, it is necessary to differentiate the underlying cardiovascular diseases as the possible causes of hemodynamic disorders or electrocardiographic changes including AVJR¹² or asystole.

Conclusion

The present case suggests that the asystole, which is associated with VVR triggered by venipuncture for peripheral intravenous catheterization in a patient with dental anxiety involved in lower pain threshold, is a possible serious cardiac event derived from a relative imbalance of autonomic nerve activities based on painful stress. During the "lead time" from exposure to psychosomatic stressors to the onset of VVR, it is of utmost importance to detect some signs of the ECG and perform prompt systemic management in order to accomplish satisfactory stress-free dental care and prevent the deterioration of VVR.

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Authors contributions

H.S. drafted the manuscript, and T.T. and K.Y. revised the manuscript. All authors read and approved the final manuscript.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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