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NOTE

Internal Medicine

Situational syncope caused by vomiting in a cat

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ABSTRACT. A 14-year-old spayed female mixed-breed cat weighing 3.4 kg was admitted to the Tokyo University of Agriculture and Technology Animal Medical Center for syncope after vomiting. Echocardiography, electrocardiography, and thoracic radiography revealed no abnormalities. Holter electrocardiography showed a paroxysmal high-grade atrioventricular block coinciding with the vomiting. Based on these findings, the cat was diagnosed as having situational syncope with paroxysmal high-grade atrioventricular block triggered by vomiting, which improved with antiemetic treatment alone. The cat subsequently died of chronic kidney disease approximately 3 years and 4 months after the initial diagnosis. This is the first reported case of situational syncope triggered by vomiting in a cat.

KEYWORDS: cat, high-grade atrioventricular block, neurally mediated syncope, situational syncope, vomiting

Syncope, a transient disturbance in consciousness, is caused by global cerebral ischemia [3, 8, 17, 19]. In neurally mediated syncope, a vagal reflex triggers bradyarrhythmia or hypotension, resulting in syncope [3, 4, 11]. Situational syncope, vasovagal syncope, and carotid sinus syndrome are neurally mediated types of syncope. Situational syncope is induced by specific situations or daily activities, such as urination, defecation, swallowing, or coughing [9, 11, 19]. Cardiogenic syncope, which is due to structural cardiac disease, accounts for most syncopal episodes in cats [5, 7]. To the best of our knowledge, there have been no reported cases of situational or neurally mediated syncope in cats. We herein describe a case of situational syncope due to vomiting in a cat with paroxysmal high-grade second-degree atrioventricular block.

A 14-year-old spayed female mixed-breed cat weighing 3.4 kg (body condition score: 3/5) with no relevant medical history was referred to the Tokyo University of Agriculture and Technology Veterinary Medical Center with the chief complaint of collapse and loss of consciousness after vomiting 4–5 times a day (on the 1st day). The vomiting and associated syncopal episodes began 104 days prior to the initial visit to our facility; however, the patient was referred to us owing to an increased frequency of syncope associated with vomiting. The syncopal episodes occurred only after vomiting, and the patient returned to a normal state of consciousness after approximately 10 sec. At the time of loss of consciousness, the patient went completely recumbent; tonic-clonic seizures were not observed. The patient was in good general condition at the time of the initial presentation to our facility, with a palpable femoral pulse of 156 bpm, which was within the normal range, and no pulse irregularities. Auscultation revealed no abnormalities in cardiac or respiratory sounds, and no other clinical signs suggestive of cardiac disease were noted on the physical examination. Complete blood cell counts and blood biochemical analyses were within normal limits, as were serum concentrations of feline pancreatic-specific lipase (Spec FPL; IDEXX Laboratories, Tokyo, Japan), symmetric dimethylarginine (SDMA; IDEXX Laboratories), thyroxine (T4; Fujifilm Vet Systems, Tokyo, Japan), and high-sensitivity cardiac troponin I (hs-cTnI; Fujifilm Vet Systems).

Thoracic radiographs revealed no abnormalities in the morphology or size of the heart (vertebral heart score, 7.1 vertebrae [reference range, ≤8.1]) [12] or lung field opacification. Abdominal radiography revealed no abnormalities. Echocardiography showed no obvious enlargement of the cardiac chambers, and the left atrial to aortic root ratio was 1.3, within the normal range (<1.5 [1]). End diastolic ventricular septal wall and left ventricular free wall thicknesses were 2.8 and 4.0 mm, respectively, both within normal limits [13]. No valvular abnormalities, cardiac shunts, intracardiac structures, or endocardial echogenicity were observed. Six-lead electrocardiography showed a low QRS amplitude but the patient was in sinus rhythm (Fig. 1). As the aforementioned examination results did not identify the cause of the patient's loss of consciousness, Holter electrocardiography was performed at home for 3

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days. The patient was found to have syncope following vomiting 4–5 times per day starting on the first day of Holter monitor use. In addition, it was found that paroxysmal high-grade atrioventricular block occurred at the time of the syncopal episodes (Fig. 2), when there was a gradual increase in the PP and PR intervals compared to sinus rhythm. These changes were not observed during sinus rhythm (Fig. 3). The longest QRS complex loss due to atrioventricular block occurrence was approximately 7.5 sec. No atrioventricular block was observed, except after vomiting, and no other arrhythmias were observed.

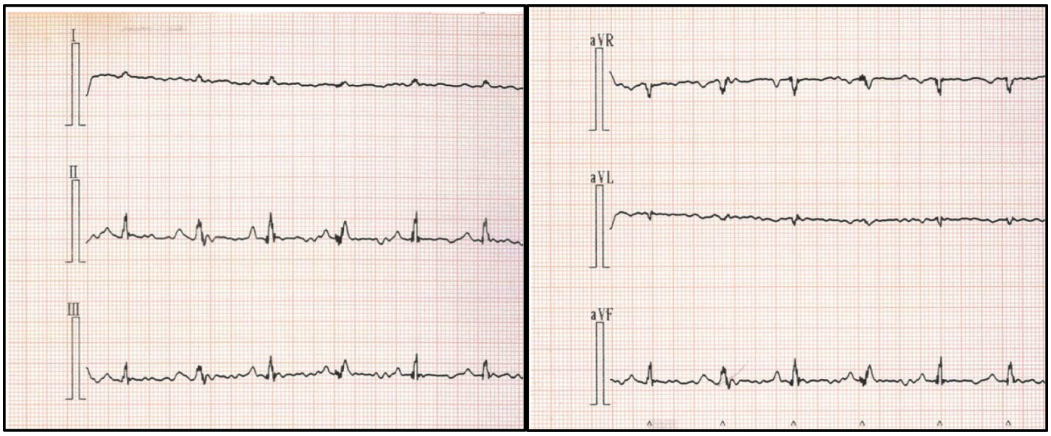

Fig. 1. Six-lead electrocardiography shows sinus rhythm with a lower QRS amplitude. Paper speed=25 mm/sec; 1 cm=0.5 mV; Heart rate (HR)=173 bpm.

Fig. 2. Holter electrocardiography of the case. Paroxysmal high-grade atrioventricular block (red frame) occurs at a timing perfectly consistent with the symptoms. White line=ventricular arrest (7.5 sec).

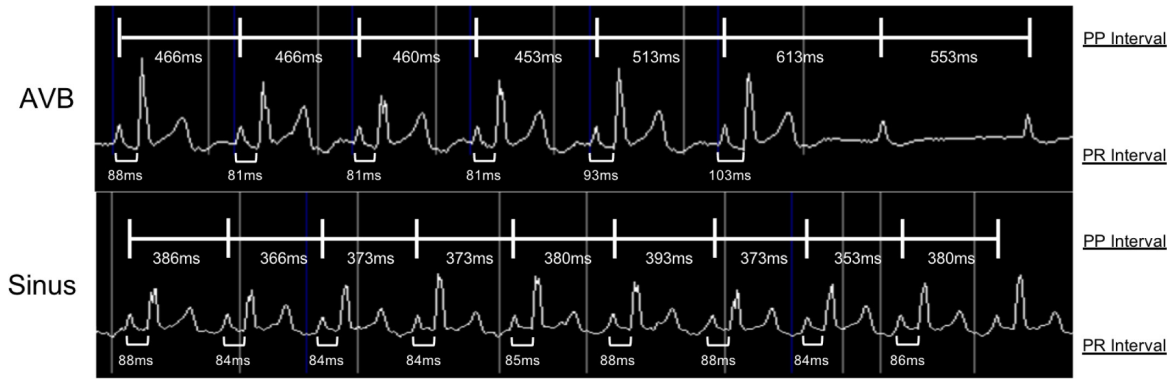

Fig. 3. Holter electrocardiography of the case. The PP and PR intervals are clearly prolonged with the appearance of atrioventricular block, suggesting that vagal tone is involved in this atrioventricular block.

As the syncopal episodes were observed only after vomiting and Holter electrocardiography showed a paroxysmal high-grade atrioventricular block consistent with the symptoms, the patient was diagnosed with situational syncope with paroxysmal high-grade atrioventricular block triggered by vomiting. No findings suggestive of gastrointestinal disease were found on examination, and the cause of the vomiting remained unclear. Antiemetic treatment with famotidine 0.8 mg/kg twice a day and metoclopramide 0.5 mg/kg twice a day resulted in marked improvement of the vomiting. In addition, the frequency of syncope was markedly reduced accordingly. Metoclopramide was discontinued after approximately 2 weeks, and only famotidine was continued. Since the start of treatment, the frequency of vomiting has decreased to approximately once every 1–2 weeks, with syncopal episodes occurring approximately once every 6 months. Approximately 624 days after the initial visit syncopal events were no longer observed. The patient died of chronic kidney disease approximately 3 years and 4 months (1,210 days) after the initial diagnosis. We were unable to perform a postmortem pathological examination because the owners' consent was not obtained.

Although, we often encounter cases of cardiogenic syncope caused by bradyarrhythmias such as complete atrioventricular and paroxysmal high-grade second-degree atrioventricular blocks, or tachyarrhythmias such as paroxysmal supraventricular tachycardia in cats, this is the first reported case of situational syncope triggered by vomiting in a cat [5, 6]. In veterinary medicine, situational syncope has only been reported in association with coughing and swallowing in dogs [10, 18]. However, there are very few reports on situational syncope caused by vomiting, even in humans [15]. The mechanism of the vagal reflex when caused by vomiting is not completely understood, although it is currently thought to be due to the increased sensitivity of baroreceptors in the esophagus [17].

In cats, paroxysmal high-grade atrioventricular block commonly occurs when there is a structural abnormality in the electrical conduction system, and the PP interval is generally unchanged or only slightly increased during the atrioventricular block [7, 14]. However, the electrocardiographic findings in this case differed from those of a typical paroxysmal high-grade atrioventricular block, as the PP interval was mildly but gradually prolonged before finally leading to a block. This variation in the PP interval only occurred in conjunction with the atrioventricular block, and was not observed during sinus rhythm. Changes were also observed in the PR interval, which was prolonged immediately prior to the atrioventricular block. A prolonged PR interval is often observed in humans and dogs with Mobitz type 1 atrioventricular block as the result of vagal tone. The aforementioned electrocardiographic features are seen in neurally mediated syncope and respiratory arrhythmias in humans and dogs, suggesting that these events are caused by changes in autonomic balance, particularly enhanced vagal tone [16, 18, 20]. The patient in this case underwent heart rate variability analysis to evaluate the autonomic nervous activity. The coefficient of variation of R-R intervals (CVRR: %) was calculated for the 30-min arrhythmia period (arrhythmia) and the 30-min before (before) and after (after) arrhythmia periods (mean ± values). CVRR data were statistically analyzed by Kruskal-Wallis test with Schefft's multiple comparison test. The obtained CVRR data were 10.8 ± 4.8% for before, 10.7 ± 3.9% for arrhythmia, and 10.2 ± 1.5% for after, respectively, with no statistically significant difference between the groups. The vagal reflex is an instantaneous change; therefore, no significant results were obtained in the evaluation of the autonomic nervous system activity during the syncopal episodes in this case. Although age-related changes in autonomic balance are common in humans, it is unclear whether similar changes occur in cats [2]. In this case, the patient also had decreased fluid volume and autonomic nervous system imbalance due to aging; therefore, vomiting could have easily triggered neurally mediated syncope.

The differential diagnosis of syncope in this case may have been vasovagal syncope in response to excessive sympathetic tone caused by vomiting. However, this diagnosis was excluded because there was no evidence of a tachycardic phase due to sympathetic tone in the preliminary phase leading to bradycardia and subsequent cardiac arrest [16, 18].

Using electrophysiological studies, situational syncope can be more reliably diagnosed by confirming the absence of abnormalities in the electrical conduction system. However, electrophysiological studies are challenging to perform in smaller dogs and cats, and electrophysiological abnormalities were not examined in this case. Additionally, the presence of electrical conduction system abnormalities such as those in the atrioventricular node and bundle of His was unconfirmed in this case, as a postmortem pathological examination could not be performed. These were some of the limitations when diagnosing this patient. In this case, the syncopal episodes were only observed after vomiting, which strongly supported the diagnosis of situational syncope due to vomiting. Additionally, the patient may have had gastrointestinal disorders; however, endoscopy could not be performed because the owner did not consent to the anesthetic procedure. Although situational syncope has received limited attention as the cause of syncope in cats, a detailed history, Holter electrocardiography can reveal the incidence, clinical presentation, and optimal treatment of situational syncope in cats.

CONFLICTS OF INTEREST. The authors have no conflicts of interest directly relevant to the content of this article.

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