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Canine case of swallowing syncope that improved after pacemaker implantation

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ABSTRACT. A 14-year-old intact male West Highland White Terrier weighing 6.9 kg was admitted to the Tokyo University of Agriculture and Technology Animal Medical Center with the complaint of syncope after showing signs of nausea during feeding. Sinus arrest induced by deglutition was confirmed using a Holter electrocardiography test. However, the clinical symptoms significantly improved after implantation of a permanent pacemaker. Seven months after implantation, the dog died from acute pancreatitis, a cause unrelated to the syncope. Immediately after its death, the heart, lungs, gastrointestinal tract, and other organs were dissected and examined histopathologically. The brain was also examined using magnetic resonance imaging. Examination results led to the diagnosis of swallowing-induced situational syncope.

KEY WORDS: dog, permanent pacemaker implant, swallowing syncope

Syncope is defined as a transient disturbance of consciousness due to global cerebral ischemia [1, 5, 10]. Neurally mediated syncope syndrome is known as a cause of syncope onset and may develop in the absence of underlying structural cardiac disorders or central nervous system disorders [3, 14, 15]. Neurally mediated syncope syndrome includes vasovagal syncope, emotional syncope, carotid sinus syndrome, and situational syncope [2, 15]. Situational syncope is defined as a syncope that is triggered by specific conditions or daily activities such as urination, defecation, swallowing, coughing, or vomiting [15, 23]. The underlying mechanism for situational syncope involves acceleration of rapid vagal nerve activity and bradycardia, blood pressure reduction caused by a decline in sympathetic nerve activity, and a reduction in cardiac preload [2, 15, 20]. In veterinary medicine, cases of canine and feline syncope caused by cardiac disorders and arrhythmias are common. However, to our knowledge, only a few case reports describe details regarding canine situational syncope [21]. We report the case of a dog with syncope triggered by swallowing; The dog's condition improved after pacemaker implantation.

This case involves a 14-year-old intact male West Highland White Terrier weighing 6.9 kg (body condition score of 3/5). The dog's medical history included chronic kidney disease (International Renal Interest Society Stage II Sub 1), and it had been treated orally with activated carbon. The dog was admitted to the University of Agriculture and Technology Animal Medical Center (TUAT-AMC) after displaying signs of syncope for one month. The onset of syncope was preceded by nausea during feeding. Syncope was observed more than three times a week, and only during feeding. However, the dog did not show unsteadiness or exercise intolerance during normal daily living. In addition, while the dog was unconscious for 3 to 5 sec, neither convulsion nor nystagmus was observed. The primary veterinarian instructed a change of diet from solid to liquid form, and suggested feeding in small amounts. The owner followed this advice. However, the frequency of syncope increased. After recovery of consciousness from syncope, the dog exhibited normal behavior. Upon physical examination, no abnormality was found via palpation, and a neurological examination did not reveal any abnormalities. Likewise, cardiac and lung auscultations did not detect any abnormal sounds. In addition, no abnormalities were found in the complete blood count. Biochemical analysis of the blood sample did not reveal any abnormalities other than mild elevations of plasma urea nitrogen, plasma creatinine, plasma alkaline phosphatase, and plasma chloride concentrations. Preprandial and postprandial total bile acid concentrations and thyroid hormone concentration

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Table 1. Values of complete blood count and biochemical analysis

WBC	7.6 × 10^3 /μl	(60–170)	GLU	130 mg/dl	(75–128)
RBC	5.94 × 10^6 /μl	(550–850)	BUN	44.3 mg/dl	(9.2–29.2)
HGB	13.7 g/dl	(12–18)	CRE	2.0 mg/dl	(0.4–1.4)
HCT	41.4%	(37–55)	ALT	61 U/l	(17–76)
MCV	69.7 fl	(60–77)	AST	61 U/l	(17–44)
MCHC	33.1 g/dl	(32–36)	ALP	351 U/l	(47–254)
PLT	254 × 10^3 /μl	(200–500)	T.BIL	0.5 mg/dl	(0.1–0.5)
TBA pre [a)]	4.8 μmol/l	(<9.0)	TP	7.5 g/dl	(5.0–7.2)
TBA post [b)]	11.8 μmol/l	(<14.9)	ALB	3.7 g/dl	(2.6–4.0)
T4	0.9 μg/dl	(0.9–4.4)	Na	148 mEq/l	(141–152)
FT4	10.2 pmol/l	(7.7–38.6)	K	4.6 mEq/l	(3.8–5.0)
TSH	0.27 ng/ml	(0.02–0.32)	Cl	119 mEq/l	(102–117)

a) Before meals, b) 2 hr after meals.

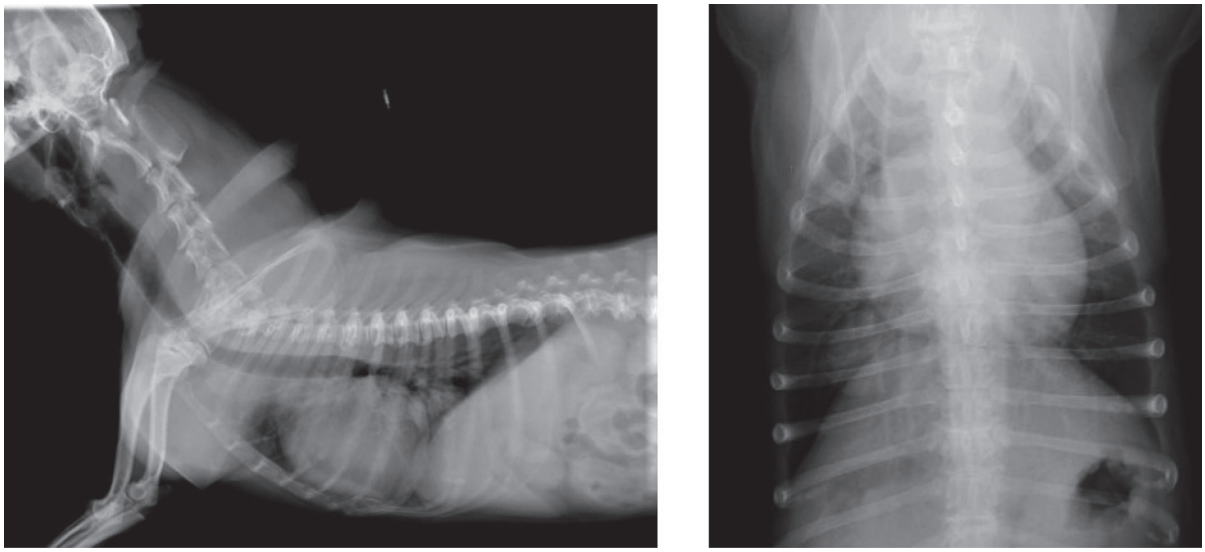

Fig. 1. Chest X-ray findings. Calcification of the bronchi and a mass on the right caudal lung lobe.

were within the reference ranges (Table 1). Chest radiography revealed calcification of the bronchi and a mass on the right posterior lung lobe (Fig. 1). The vertebral heart size was 10.1 vertebrae, which was within the reference range (<10.5) [22]. Echocardiography (Logiq7, GE Medical, Tokyo, Japan) revealed a ratio of 1.3 for the left atrium to the aorta in B-mode (reference range: <1.6) [24]. In the M-mode analysis, the left ventricular internal dimension in diastole and in systole were 29.5 mm and 16.4 mm, respectively (reference ranges: 17.8–29.6 mm and 9.9–19.8 mm, respectively). The fractional shortening obtained from these values was 44.4% (35–45%) [4]. Doppler-mode analysis showed an early diastolic filling velocity of 73.5 cm/sec (91 ± 15 cm/sec) and an atrial filling velocity of 50.1 cm/sec (63 ± 13 cm/sec) [11]. All of the analyses revealed measurement values within their reference ranges. The cardiac index was 4.9 l/ min/m^2, which was slightly accelerated (3.1–4.7 l/ min/m^2) [12]. No morphological abnormalities such as atrioventricular valve regurgitation or cardiac defects were observed. In addition, abdominal ultrasonography did not yield any abnormal findings. Blood pressure was measured using an indirect blood pressure measuring instrument for animals (BP100D, Fukuda M.E Kogyo Co., Tokyo, Japan), which revealed systolic, mean, and diastolic blood pressures of 145, 114 and 97 mm Hg, respectively, all of which were within their reference ranges [6]. Scalar electrocardiography (D300, Fukuda M.E Kogyo Co.) revealed a heart rate of approximately 100 bpm in sinus rhythm and no abnormalities other than mild elevation of the R wave. An atropine loading test was performed with intravenous administration of atropine sulfate 0.5 mg (Atropine Sulfate Injection, Mitsubishi Tanabe Pharma, Osaka, Japan). The heart rate increased from 100 bpm prior to the loading test to 200 bpm after the loading test. In addition, the heart rate remained higher than 160 bpm after 15 min from the time of the atropine sulfate administration. From the results of the examinations, the cause of the syncope could not be identified from the results of these examinations. Therefore, to determine the cause, a Holter electrocardiography (ECG) examination (QR2500, Fukuda M.E Kogyo Co.) was performed at home, and the owner was asked to maintain a detailed record of the animal's activities such as feeding, defecation, urination, walking, and sleeping. This revealed the occurrence of syncope during feeding. Analysis using specialized software (HS1000, Fukuda E.M Kogyo Co.) revealed recordings of sinus arrest lasting more than 11 sec, which matched the feeding time recorded by the owner (Fig. 2). Thus, the dog was diagnosed as having swallowing-induced situational syncope. As

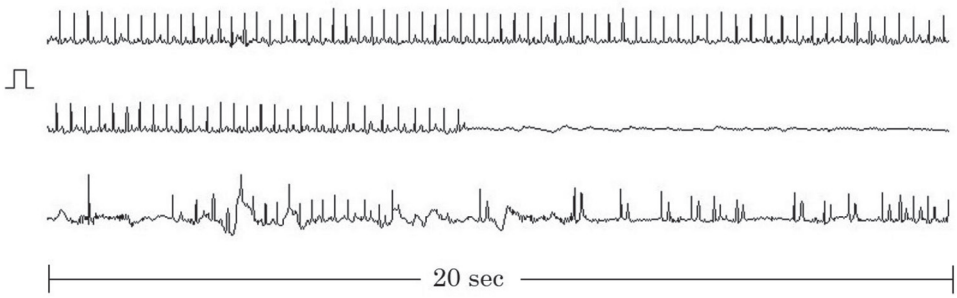

Fig. 2. Holter electrocardiographic findings. Holter electrocardiography revealed the occurrence of syncope during feeding. Analysis showed recordings of sinus arrest lasting more than 11 sec, which matched the feeding time recorded by the owner. Recorded by MX-lead.

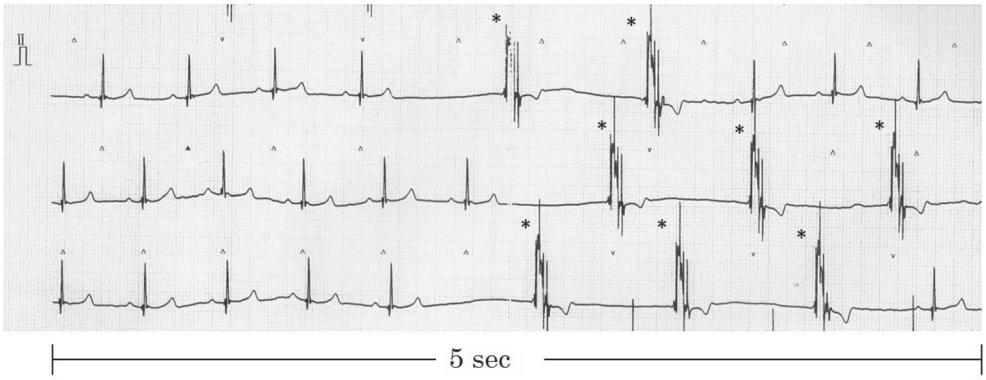

Fig. 3. Electrocardiographic findings after pacemaker implantation. Syncope was no longer observed after pacemaker implantation; nevertheless, the heart stopped beating during feeding. Pacemaker activity was confirmed during feeding. Recorded by II-lead. Paper speed 50 mm/sec. *: QRS complex formed by pacemaker stimulation.

treatment, a permanent pacemaker was implanted. A pre-anesthetic procedure involving intravenous injection of cefazolin 25 mg/kg (Cefamezin α, Astellas, Tokyo, Japan) was performed; an atropine sulfate injection at 0.05 mg/kg (Atropine Sulfate Injection 0.5 mg, Mitsubishi Tanabe Pharma) was administered 5 min later, an intravenous injection of midazolam was administered at a dose of 0.2 mg/kg (Dormicum, Astellas), and butorphanol tartrate was administered at a dose of 0.2 mg/kg (Vetorphale, Meiji Seika Pharma, Tokyo, Japan). Anesthetic induction was achieved by slow intravenous administration of propofol at a dose of 3 mg/kg. Anesthesia was maintained with an inhalation of 100% oxygen and 1 to 2% isoflurane. Ventilation was manually controlled during the surgery. An incision was made from the left jugular vein, and an electrode lead (CapSureFix NOVUS, Medtronic Inc., Minneapolis, MN, U.S.A.) 58 cm long was inserted from the incision site. The electrode tip was placed within the right ventricle and connected to a generator (Adapta DR, Medtronic Inc.; stimulus sensing model VVI, basic rate of 80 bpm, stimulus threshold of 3.5 mV, and pulse width of 0.4 msec) embedded in the subcutaneous pocket at the neck. In addition, to prevent thrombus formation, dipyridamole 12.5 mg/head was prescribed to be taken orally twice a day after the surgery. Syncope was no longer observed after the pacemaker implantation; nevertheless, the heart stopped beating during feeding, and pacemaker activity was confirmed during feeding (Fig. 3). For 7 months after the pacemaker implantation, no swallowing-induced syncope occurred, and the clinical course during that period was favorable. However, the dog was fed a large amount of high-fat food during a party hosted by the owner, and 2 days later, the dog was examined by the veterinarian because of symptoms including exhaustion, lack of appetite, frequent vomiting, and diarrhea. The dog did not respond to the palliative therapy administered for a week by the veterinarian, and the clinical symptoms worsened; thus, the dog was taken to TUAT-AMC. When the dog was examined at the animal medical center, its body temperature was 39.5°C, and symptoms of panting and abdominal tenderness were observed. The dog also vomited during the clinical examination. Abdominal ultrasonography was indicative of gastric ileus, and an increased signal intensity was observed in the pancreas. The white blood cell count ($52.1 \times 10^3/\mu l$) and pancreas-specific lipase value (1,000 $\mu g/l$; ≤200 $\mu g/l$) were extremely high. Moreover, the blood urea nitrogen level was 85.0 mg/d*l*, the creatinine level was 4.5 mg/d*l*, and the urine volume was decreased. Based on these data, the dog was diagnosed with acute pancreatitis, which in turn caused exacerbation of renal failure. The day after the dog was presented at our institution, the dog stopped producing urine and died. With the owner's consent, the pacemaker was removed immediately after death, and pathological examinations of the heart, lung, and abdominal organs were performed. Simultaneously, magnetic resonance imaging of the head (MRI; AIRIS II, Hitachi Medical Corp., Tokyo, Japan) was also performed. MRI of the brain did not reveal abnormalities in the T1-weighted image, the T2-

weighted image, or the fluid-attenuated inversion recovery image. During the necropsy, a microscopic examination of the heart and the conducting system (sinoatrial node, atrioventricular node, and His bundle) revealed no remarkable pathological changes. From these findings and the fact that no pathological changes were observed in the brain or heart, it was determined that the syncope was caused by swallowing-induced situational syncope. Macroscopic lesions were observed in the right caudal lung lobe, pancreas, and lower part of the esophagus. Irregularly shaped and poorly demarcated masses were revealed in the right caudal lung lobe and diagnosed as pulmonary adenocarcinoma. The pancreas was slightly swollen from diffuse interlobular edema with small hemorrhagic foci. The most prominent tissue alterations in the pancreas were acinar cell necrosis, an intense acute inflammatory reaction, and foci of necrotic fat cells, which are consistent with acute pancreatitis. Mild to moderate catarrh and congestion were found in the esophagus and were considered relatively newly developed lesions. These findings are consistent with those in many cases of reflux esophagitis. The lesions may have developed secondarily to acute pancreatitis. The results of the necropsy suggest that acute pancreatitis was the cause of the animal's death. However, the underlying conditions that resulted in the onset of swallowing syncope remain unclear.

Diagnosis of situational syncope in humans requires an understanding of the situation at the time of syncope. This understanding is gained from detailed medical history and elimination of any disorders or conditions that can cause syncope, such as cardiovascular, neurological, and metabolic disorders [1, 9, 13]. Similarly, an understanding of the situation at the time of syncope as revealed by the owners and elimination of any underlying diseases known to cause syncope are the most important steps for the diagnosis of situational syncope in dogs. Furthermore, the Holter ECG test, which is conducted while the owners record the activity of the animal, is considered a highly efficient diagnostic test. The Holter ECG test was useful in determining an antemortem diagnosis in the present case.

Stimulation of the receptors specific to each clinical condition, that is, esophageal receptors with increased sensitivity for swallowing-induced syncope [17, 18, 20], is transmitted to the nucleus tractus solitarius of the medulla oblongata, which in turn stimulates the nucleus of the vagus nerve in this area and/or suppresses the vasomotor center, thereby causing situational syncope [9, 13]. Therefore, syncope may be classified into three types: 1) cardiac-suppression type, where bradycardia or cardiac asystole is induced; 2) vasodepression type, where only blood pressure is reduced; and 3) mixed type, where both heart rate and blood pressure are reduced [13]. Among these, the cardiac suppression and mixed types can be easily diagnosed in dogs using the Holter ECG test to detect bradycardia or cardiac asystole. However, diagnosis of the vasodepression type is not easy, as it is extremely difficult to observe the hypotension that causes the syncope. Sinus arrest consistent with deglutition was confirmed on the Holter ECG test in the present case, and a diagnosis of swallowing-induced syncope of the cardiac suppression or mixed type was made. Moreover, cardiac disorders and neurological disorders were excluded through other tests, which further supported the diagnosis.

In humans, concurrent esophageal diseases such as esophageal hernia, esophageal spasm, diverticulum, cancer, and achalasia are observed in 38.75% of patients with swallowing-induced syncope [16, 18, 21, 23]. However, a previous report indicates that in 38.75% of patients, no underlying cause that may increase the sensitivity of the esophageal pressoreceptor is present or identifiable [18]. In the present case, esophagitis, one of the symptoms of acute pancreatitis, was observed and was associated with vomiting. However, as the formation of this lesion was relatively new, the causal relationship between esophagitis and swallowing-induced syncope was considered remote. Moreover, in the present case, formation of lung adenocarcinoma was observed in the posterior lobe of the right lung. Neoplastic lesions in the lung can compress the esophagus, which may cause swallowing-induced syncope [8, 10]. However, in the present case, as the lung lesion was not in contact with the esophagus, this lesion probably did not affect the esophagus. Thus, no concurrent disease that could cause swallowing-induced syncope was identified.

Currently, the treatment protocol for situational syncope in dogs has not been established. In Japan, the treatment guideline for situational syncope for humans is stipulated in the Guidelines for Diagnosis and Treatment of Syncope (JCS 2007 and 2012) [9, 13]. According to these guidelines, class 1 (benefits are evident and its applicability is generally agreed) includes the patient's understanding of the clinical condition, lifestyle guidance, and avoidance of syncope at the time of the appearance of prodromal symptoms [9, 13]. Lifestyle guidance for swallowing syncope includes avoidance of contributory solids, hot water, cold water, and carbonated drinks, as well as swallowing solids after sufficient chewing [9, 13]. Furthermore, class IIa (largely agreed as beneficial) includes pacemaker implantation for severe cases and the cardiac-suppression type [9, 13]. With respect to class I of the human guidelines, in veterinary medicine, the owner can only implement lifestyle restrictions for the dog. In the present case, the owner implemented these lifestyle changes and medical treatments for the dog, but it was not enough to prevent syncope. Considering that pacemaker implantation has proven effective in swallowing-induced syncope in humans [7], pacemaker implantation was chosen for the treatment of this dog. A favorable outcome equivalent to that observed in humans was obtained after pacemaker implantation. Thus, pacemaker implantation is considered one of the effective therapies for swallowing-induced syncope in dogs.

In the treatment of cardiac-suppression and mixed types, atropine sulfate, which is an anticholinergic drug, causes blocking of the vagus nerve, and its administration is theoretically effective. Bradycardia during swallowing was avoided by the administration of atropine sulfate in this case. However, oral administration of atropine sulfate is difficult for several reasons, including strong side effects such as tachycardia, dry mouth, photophobia, and constipation. An oral form of the drug does not exist, and the injectable form is bitter in taste. Hence, it is not suitable as a long-term therapeutic agent to maintain clinical conditions. Furthermore, the effects of other anticholinergic drugs on the cardiac vagal nerve are weak [9, 13]. Recently, reports have shown that cilostazol, an antiplatelet drug, is effective in the treatment of bradyarrhythmia in humans [19] and dogs [14]. In some cases of situational syncope in animals, the owners do not agree to pacemaker implantation or the animal has complications that make implantation impossible. Therefore, medical treatment using drugs such as cilostazol should be further investigated.

Presently, situational syncope as the cause of syncope in dogs has received limited attention. However, detailed clinical history,

Holter ECG tests, and echocardiography could better reveal the frequency of situational syncope in dogs, the animals' clinical conditions, and the best course of treatment.

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