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<Case Report>

Atropine-induced atrial bigeminy during general anesthesia in a Cocker Spaniel dog

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Abstract: A 12-year-old female Cocker Spaniel (7.5 kg of body weight) was presented for resection of a mammary gland tumor. During surgery, the heart rate was remarkably decreased due to a second-degree type I atrioventricular block. Atropine (0.05 mg/kg) was administered to increase the heart rate. Although the heart rate was elevated, atrial bigeminy occurred and persisted until the dog fully recovered from general anesthesia. These results highlight the possibility of atrial bigeminy caused by atropine administration during anesthesia.

Keywords: atrial bigeminy, atrial premature complex, atropine, dog, sinus bradycardia

Atrial bigeminy is a type of atrial arrhythmia consisting of the repetitive sequence of one atrial premature complex (APC) followed by one sinus beat [2]. Although ventricular bigeminy (ventricular premature complex followed by sinus beat) is more familiar and important in clinical situation, atrial bigeminy should have attention in dogs with heart diseases or conditions linked to the occurrence of APCs [4]. This case report described atrial bigeminy occurred after atropine administration during general anesthesia induced with diazepam, butorphanol and propofol, and maintained with isoflurane.

A 12-year-old female Cocker Spaniel (7.5 kg of body weight) was presented with a mammary gland tumor. Because cytological evaluation revealed malignancy on mammary gland cells, total mastectomy with ovarian hysterectomy was decided. Before surgery, pre-anesthetic tests were performed. Physical examination found no particular abnormalities except poor body condition score (2/5) and dehydration. Systolic blood pressure measured by Model 811-B Doppler Ultrasonic Flow Detector (Parks Medical Electronics, USA) was 130 mmHg. Electrocardiogram (ECG) studies revealed normal sinus rhythm with ~120 beats/min. Complete blood cell count and serum chemistry profiles revealed upper normal limit of total protein (7.0 g/dL; reference range: 5–7.2 g/dL) increased hematocrit (58.2%; reference range: 37–55%), increased sodium (149 mEq/L; reference range: 138–148 mEq/L) and increased chloride (122 mEq/L; reference range: 102–118 mEq/L), suggesting dehydration. Thoracic radiography found no cardiomegaly (vertebral heart scale 10.2) and no pulmonary metastasis. Surgical anesthesia was achieved with diazepam (0.5 mg/kg, intravenously [IV], Valium; Myung-In Pharm, Korea) and butorphanol (0.1 mg/kg, IV; Myungmoon Pharm, Korea) premedication, propofol (2.5 mg/kg, IV, Propofol; Myungmoon Pharm, Korea; titrated to effect) induction and isoflurane (1–5%, Ifran; Hana Pharm, Korea) maintenance. Thirty minutes after surgery, the heart rate was remarkably decreased (~60 beats per min) with occurrence of second degree type I atrioventricular (AV) blocks. Atropine (0.05 mg/kg, IV, Atropine sulfate injection; Jeil Pharmaceutical, Korea) was administered to abolish AV blocks and to increase the heart rate. ECG tracing from patient monitor found every other beat had atrial ectopic beats that were slightly different from the preceding sinus beats, although ventricular beats were not significantly different (Fig. 1). However, the waveforms from pulse oximetry indicated no pulsation after ventricular beats from abnormal atrial beats. ECG tracing from 3-lead surface ECG recorder also revealed an APC follows every SB (atrial bigeminy; Fig. 1). The P-waves from APC were often buried with T-waves from proceeded sinus beat. Despite this abnormal rhythm, there was no significant reduction in SpO_2, oxygen saturation by pulse oximetry (~96%) and mean aortic pressure (~80–90 mmHg). Surgery was successfully finished without any medical intervention. The atrial bigenimy was persisted until the dog was fully recovered from general anesthesia.

In human, the ectopic activity is increased in the atrium as the basic sinus rate is slow to near ventricular rate [1]. One

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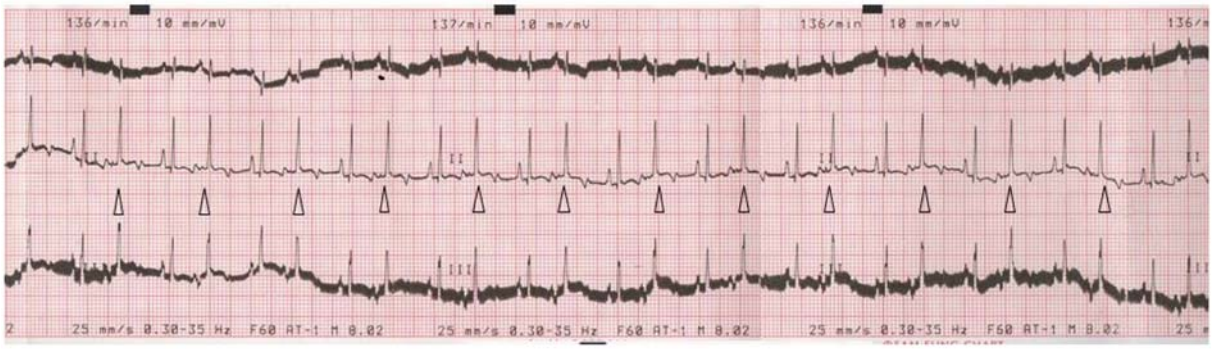

Fig. 1. The 3-lead surface electrocardiogram (ECG) taken from this case revealed abnormal atrial premature contractions (APC; arrow heads) followed by every sinus beats (atrial bigeminy). The P-waves from APC were often buried with T-waves from proceeded sinus beat. Although the heart rate was 130–140 bpm on the ECG, the actual pulsation rate was 65–70 bpm on pulse oximetry, because of atrial bigenimy.

study suggested the mechanisms of ectopic impulse formation in this situation might be due to focal re-excitation of already repolarized fibers by neighboring fibers still depolarizing [1]. Vagal stimulation can also reduce refractory period in the atrium and thus can increase the asynchrony of recovery of excitability in the atrium. Both mechanisms might be involved in occurrence of atrial ectopy in bradycardic patients [1]. Therefore, the administration of atropine could be useful to prevent occurrence of atrial ectopy in bradycardic dogs. However, in the present case, the atrial ectopy started to occur after administration of atropine. Although atrial bigeminy has been reported in humans taking atropine in social media, there has not yet been reported in scientific journals. Therefore, the exact cause of atrial bigeminy in the present case was unclear. The dog was markedly bradycardic (~60 beats/min) with occurrence of second degree AV blocks 30 min after general anesthesia. Therefore, vagal stimulation by anesthetic drugs was suspected, although our expectation was that heart rate might increase with abolishment of AV blocks. Although the AV blocks were abolished, the heart rate was not changed because of occurrence of atrial ectopy. Although the ECG counted ~120 beats/min, the actual pulse rate on pulse oximetry remained ~60 beats/min, suggesting those atrial ectopic beats might be pulseless. Even though atrial ectopic beats were conducted to the ventricle, there might be electromechanical dissociation causing pulseless beats. Although the dose of atropine (0.05 mg/kg) used in this case was slight higher than recommended dose (0.04 mg/kg), authors believed that this might not the cause of the occurrence of atrial bigenimy, because the higher dose of atropine was often used in dogs under cardiopulmonary resuscitation.

Veterinary reference indicated that diazepam (0.5 mg/kg) did not alter the heart rate, although butorphanol given (IV) at dose levels of 0.1 mg/kg could decrease the heart rate [3]. Inhalation anesthetics including isoflurane increase the heart rate by decreasing cardiac vagal activity in dogs [3], although it can reversely decrease the heart rate depending on the concentration used. Halothane-epinephrine-induced cardiac arrhythmias including atrial bigeminy have been reported in human [5]. In dogs under general anesthesia with propofol induction, mean the heart rate tended to increase in the first 30 min above the pre-induction values [3]. Therefore, the cause of atrial ectopy in this dog might be over-suppression of vagal tone by atropine administration. Over-suppression of vagal tone might increase sympathetic tone and thus abnormal sympathetic stimulation caused the occurrence of atrial ectopy in this dog. Since some atrial tachycardias are catecholamine sensitive, abnormally high sympathetic tone could cause atrial ectopy [2].

Although atrial ectopic beats are critical in patients with pre-existing heart diseases (by initiating atrial tachyarrhythmias and seriously compromising cardiac output), atrial bigeminy is a harmless rhythm in healthy patients [1]. However, more concrete investigation for pre-existing conditions linked to the occurrence of atrial ectopy is necessary for preventing not to proceed to malignant tachyarrhythmias.

In conclusion, this case report described atrial bigeminy occurred after atropine administration during general anesthesia. Although atrial bigeminy did not affect circulation during anesthesia, more care should be taken for not over-suppressing vagal tone by administration of atropine. These results highlight the possibility of atrial bigeminy caused by atropine administration during anesthesia.

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