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Coronary artery vasospasms in a *microminipig* occurred after placing an ameroid constrictor

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**ABSTRACT.** A 12-month-old *microminipig*, weighing 12.6 kg, showed 3 repeated episodes of transient ST-segment elevation in 24 hr Holter electrocardiogram after placing an ameroid constrictor around the left anterior descending coronary artery. Ventricular fibrillation was noticed just after the cessation of the 24 hr Holter-electrocardiogram recording. Direct current defibrillations and cardiopulmonary resuscitation were performed; however, they were unsuccessful, leading to the animal’s death. Its heart was excised for macroscopic analysis, which indicated that lumen of the ameroid constrictor was not narrowed and that there was no dissection, embolus or thrombus in the coronary arteries, indirectly suggesting that coronary artery vasospasm may have caused the ischemic attacks. Thus, *microminipig* may possess some potential to have coronary vasospasm.

**KEY WORDS:** ameroid constrictor, coronary vasospasm, sudden cardiac death

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This stretching by the retract-O-tape was carefully performed for 5 min so as not to induce the lethal ventricular arrhythmia during mounting of the ameroid constrictor (COR-1.50-SS, Research Instruments SW, Escondido, CA, U.S.A.). Thirty min later, an ameroid constrictor was placed on the left anterior descending coronary artery, and then, the chest was closed in the standard process. Fluid absorption by the constrictor causes gradual, predictable narrowing of the coronary lumen and possibly completes vessel occlusion [4].

After the chest was closed, Holter electrocardiogram was recorded from 16:07 for 24 hr. Then, the microminipig was transferred to the cage. During the whole operation, lethal ventricular arrhythmia or cardiohemodynamic collapse was not observed, except for a transit ST-segment elevation during the ischemic preconditioning. After finishing the recording of Holter electrocardiogram at 16:07 on the next day of the operation, we fed the microminipig at 17:00, and the animal suddenly fell down while eating bait. A-B lead electrocardiogram showed ventricular fibrillation. The direct current defibrillation of 360 J was applied 3 times, but could not terminate it. Cardiopulmonary resuscitation was performed, but it was unsuccessful. About 10 min later, its death was confirmed by electrocardiogram.

The Holter electrocardiogram was analyzed in the M-X leads. Three attacks of transient ST-segment elevation were recorded from 13:06:17 to 13:09:45, which lasted for 5-8 min. In the first and second attacks, tall, symmetrical and peaked T waves were initially observed, which were followed by a progressive ST-segment elevation together with the onset of premature ventricular complexes. The first and second attacks lasted for 5 and 8 min, respectively (Figs. 1 and 2). In the third attack, the similar changes in the electrocardiogram were observed to those in the first and second attacks, which lasted for 7 min; moreover, nonsustained polymorphic ventricular tachycardia occurred at a period of peak ST-segment elevation (Fig. 3).

An autopsy was performed, and the heart was excised. Macroscopic analysis indicated that there was no dissection, embolus or thrombus in the right or left coronary artery. In addition, a guidewire in a diameter of 0.035 inch was easily passed through the left anterior descending coronary artery (Fig. 4A), indicating the lumen of ameroid constrictor was not narrowed. Microscopic pathological observation was not performed, because of lack of abnormal macroscopic findings in coronary arteries and ventricular walls.

In this case, nonsustained ST-segment elevations were repeated 3 times within a few min, suggesting that events of transient and reversible transmural ischemia were induced. Several etiologies have been described that can explain the onset of transmural ischemia, i.e. coronary artery occlusion by embolism, dissection, thrombus and/or coronary vasospasm, etc. [1, 2, 7]. In this case, the lumen of ameroid constrictor was not narrowed, whereas embolism, dissection or thrombus-induced coronary artery occlusion was not identified by macroscopic pathological observation of the heart, suggesting a high possibility that the coronary vasospasm, rather than its morphological occlusions of the coronary artery caused the ischemic attacks. The pathophysiology of coronary artery vasospasm includes an increased vasomotor tone with regional sympathetic dysinnervation, endothelial dysfunction and/or increased platelet activation [3]. In this study, the mounting procedure of ameroid constrictor around the coronary artery might have provoked the endothelial dysfunction. Thus, microminipig may possess some potential to have coronary vasospasm. Experiment is now ongoing.
to demonstrate the occurrence of coronary vasospasm by using the coronary angiography with pharmacological and pathological interventions.

CONFLICT OF INTEREST. The authors declare no conflict of interest.

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Fig. 4. Photos of excised heart. (A) The heart was cut just distal to the mounting position of ameroid constrictor. A guidewire in a diameter of 0.035 inch was placed in the left anterior descending coronary artery, which was mounted by ameroid constrictor. (B) A guidewire in a diameter of 0.035 inch was placed in the left anterior descending coronary artery. The passage of the guidewire indicates the patency of the left anterior descending coronary artery. (C) Lumen of ameroid constrictor was not narrowed. RV: right ventricle; LV: left ventricle; and LAD: left anterior descending artery.

REFERENCES


