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Case Report
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Echocardiographic features of accessory mitral valve tissue presenting left ventricular outflow tract obstruction in a dog

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ABSTRACT

In a 3-year-old Samoyed, aortic bulging was found on radiography during a general check-up. On echocardiography, turbulent flow was found in left ventricular outflow tract (LVOT) with high velocity (6.1 m/s). A linear structure was attached to the interventricular septum and connected to the chordae tendineae reaching the papillary muscle. A part of the structure moved during cardiac cycle, similar to mitral motion. This dog was diagnosed with LVOT obstruction caused by accessory mitral valve tissue (AMVT). This is the first report of AMVT in veterinary medicine. AMVT should be considered as a possible cause of LVOT obstruction in dogs.

Keywords: Chordae tendineae; dog; echocardiography; mitral valve; papillary muscle

INTRODUCTION

Accessory mitral valve tissue (AMVT) is a rare congenital cardiac anomaly and refers to an accessory tissue associated with the mitral valve (MV) or MV-related apparatus [1]. AMVT may be the failure of complete separation of MV from the endocardial cushion [2,3]. Echocardiography is used as the gold standard for diagnosing AMVT in humans and allows for the detection and classification of AMVT and the determination of hemodynamic changes related to accessory tissue [2,4]. AMVT has various features according to the shape, mobility, and site of attachment of the accessory tissue [1,2,4-6]. In some cases, the appearance of AMVT may change during the cardiac cycle [4]. The AMVT is usually attached to the ventricular side of anterior MV and its vicinities [7]. Some are attached to the papillary muscle, interventricular septum (IVS), or rarely to the posterior MV [2]. AMVT usually moves according to cardiac cycles along with the MV; however, it can be paradoxical or fixed [1,5].

Some AMVT cases have features of left ventricular outflow tract (LVOT) obstruction [2]. AMVT can induce LVOT obstruction via both the mass effect of AMVT itself and complications secondary to AMVT. It includes fibrous deposition, thrombus, or remodeling of the cardiac muscle due to turbulent flow, pressure overload, and damage to cardiac muscle [3,7,8]. Therefore, when evidence of LVOT obstruction is found, AMVT should be considered as one of the underlying lesions and differentiated from more commonly identified lesions such as aortic stenosis, abnormal papillary muscle insertion, and LV false tendon [1,2].

Conflict of Interest
The authors declare no conflict of interest.

Author Contributions
Conceptualization: Choi J; Data curation: Lee SK, Choi J; Formal analysis: Kim S, Lee SK, Choi J; Funding acquisition: Choi J; Investigation: Kim S, Lee SK; Methodology: Lee SK, Choi J; Project administration: Choi J; Software: Lee SK, Choi J; Supervision: Choi J; Validation: Kim S, Lee SK, Choi J; Visualization: Lee SK, Choi J; Writing - original draft: Kim S; Writing - review & editing: Kim S, Lee SK, Choi J.

Based on our review of the literature, there is no report regarding AMVT in veterinary medicine. Therefore, this report aims to describe echocardiographic findings of AMVT in a dog and to demonstrate differentiation from other causes of LVOT obstruction.

CASE PRESENTATION

A 3-year-old neutered male Samoyed was presented because of aortic bulging on thoracic radiography at a local animal clinic during a general check-up. The dog had a history of coughing and occasional respiratory distress. The animal's blood pressure was 150 mmHg. The results of complete blood count and serum chemistry were within normal ranges. Thoracic radiography using digital radiographic system (EVA-HF525; Gemss-Medical, Korea) revealed severe bulging of the aorta (**Fig. 1**). On echocardiography using ultrasound machines (Prosound α7 Aloka; Hitach Aloka Medical, Japan), an abnormal linear structure, 11 mm in length, was attached to the IVS just below the aortic valve (AV) on the right parasternal long axis LVOT view (**Fig. 2**). This structure was connected to the chordae tendineae reaching the papillary muscle (PM). The structure shared the same PM with anterior MV and was mobile during the cardiac cycle, similar to the motion of MV. However, the base part of the structure, attached to IVS, did not move, and appeared like a rigid tissue in the subaortic region. Both PMs were found in the normal position and had normal appearance.

Both the anterior and posterior MV leaflets had a normal appearance (**Fig. 3**). However, in mid-systole, the anterior leaflet showed systolic anterior motion (SAM), causing partial LVOT obstruction. In mid-diastole, the MV leaflets drifted together toward the LV. There was no evidence of mitral regurgitation (MR). Other congenital cardiac anomalies or potential causes of LVOT obstruction were not found in any cardiac cycle.

The aortic valves had a normal linear appearance and movement. There was no dilation of the aorta. A turbulent flow below the aorta and aortic insufficiency were found on the left apical LVOT view in the late systolic phase, and LVOT velocity was severely elevated (6.1 m/s). Since the systolic pressure of the dog was 150 mmHg, the estimated systolic pressure of the LV was 300 mmHg, presenting LVOT obstruction.

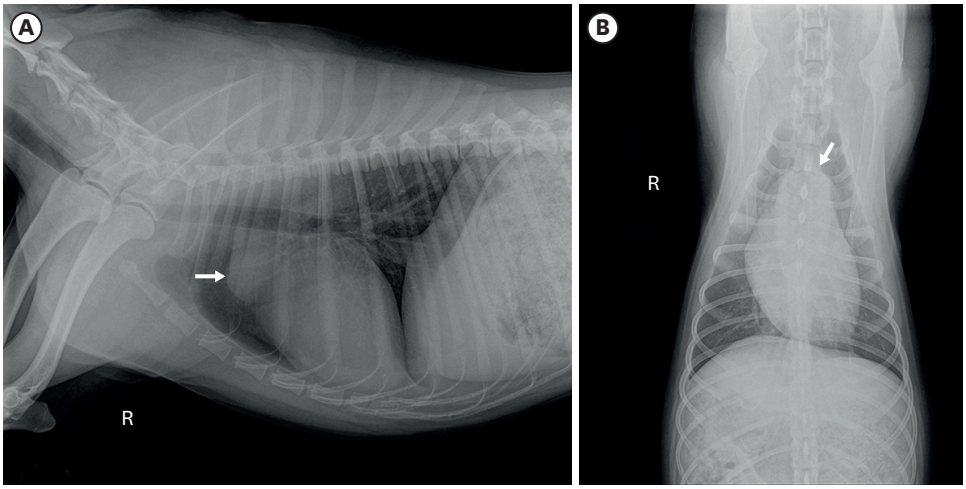

Fig. 1. Thoracic radiography. (A) Right lateral and (B) ventrodorsal views. Severe bulging of the aorta (white arrow) is seen. The shape and size of the heart are normal. There is no pulmonary infiltrate.

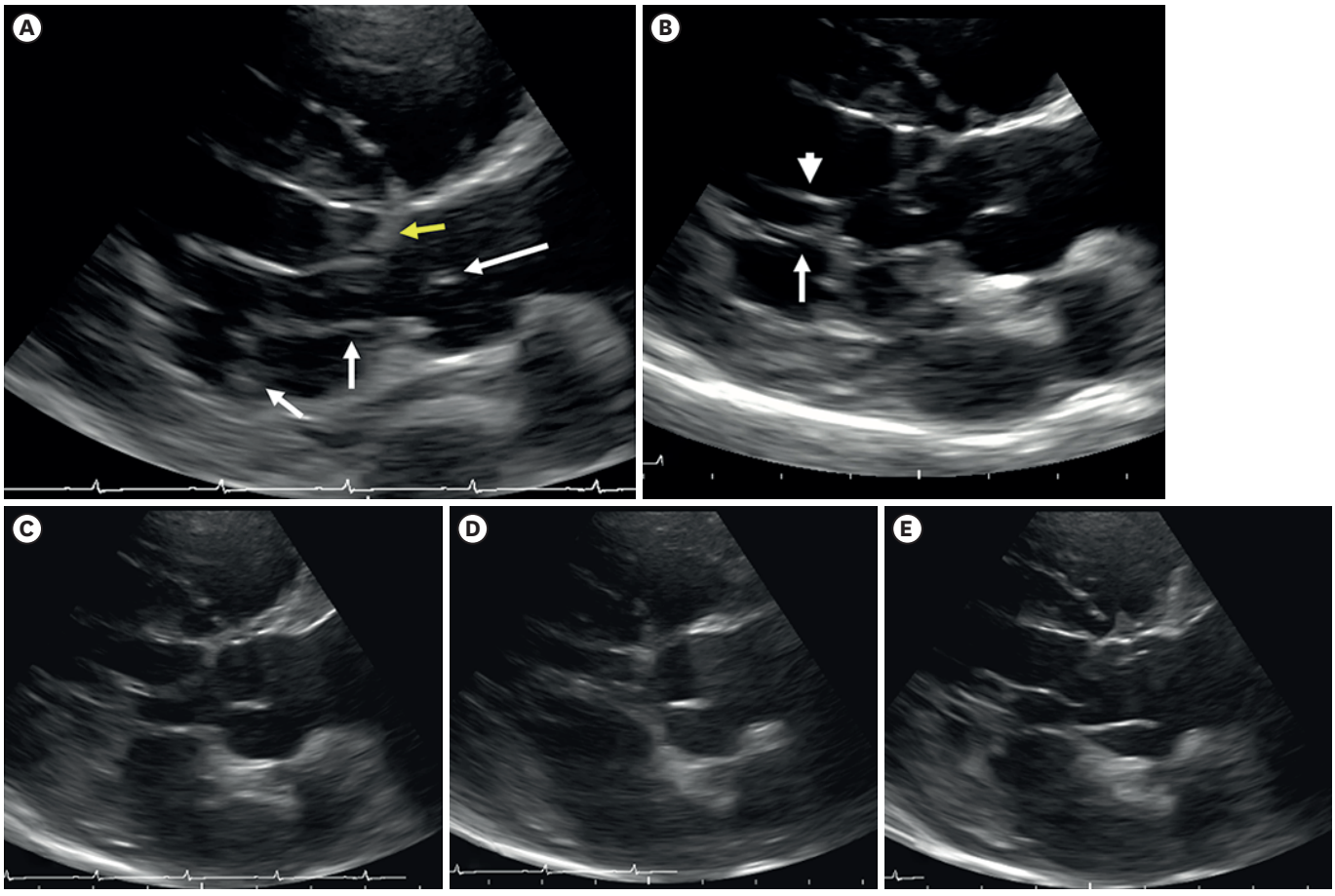

Fig. 2. Echocardiography of right parasternal LVOT view. (A) An abnormal linear structure (yellow arrow), 11 mm in length, attached to the IVS. It is located below the aortic valve (long white arrow). Both fixed and mobile portions are visualized. The white arrows indicate the mitral valve. (B) The linear structure from (A) is connected to the chordae tendineae (arrowhead), which share the same papillary muscle with the chorda of the anterior mitral valve (short white arrow). Note the movement of AMVT between the diastolic and systolic phases (C), and during diastolic (D) and systolic (E) phases.
LVOT, left ventricular outflow tract; IVS, interventricular septum; AMVT, accessory mitral valve tissue.

On M mode, flutter of both MV leaflets was observed in diastole. Since the left atrium (LA) to aorta (Ao) ratio of was within the normal range (LA/Ao = 1.3), the size of the LA was within the normal range. The thickness of IVS and LV wall was 12.5 and 14.5 mm, respectively, indicating mild thickening. The systolic function was considered normal based on the systolic left ventricular internal dimension.

The echocardiographic findings indicated that the abnormal linear structure below the AV was an accessory tissue rather than a dysplastic tissue of the MV or AV since there was no change in the MV and AV structures. The accessory tissue was located at LVOT, fixed to IVS, and showed connection to the chordae of the anterior MV. The final diagnosis was LVOT obstruction by both SAM of the anterior MV leaflet and AMVT.

The dog was discharged after prescribing atenolol (Atenol; KyungDong Pharm, Korea) 0.25 mg/kg per os (PO) twice a day (BID), clopidogrel (Plavix; Sanofi-Aventis, USA) 2 mg/kg PO SID, furosemide (Lasix; Handok Pharm, Korea) 1 mg/kg PO BID, and ramipril (Ramipril Tab; Sandoz, Korea) 0.125 mg/kg PO SID was administered for 7 days. Unfortunately, the dog was lost to follow-up.

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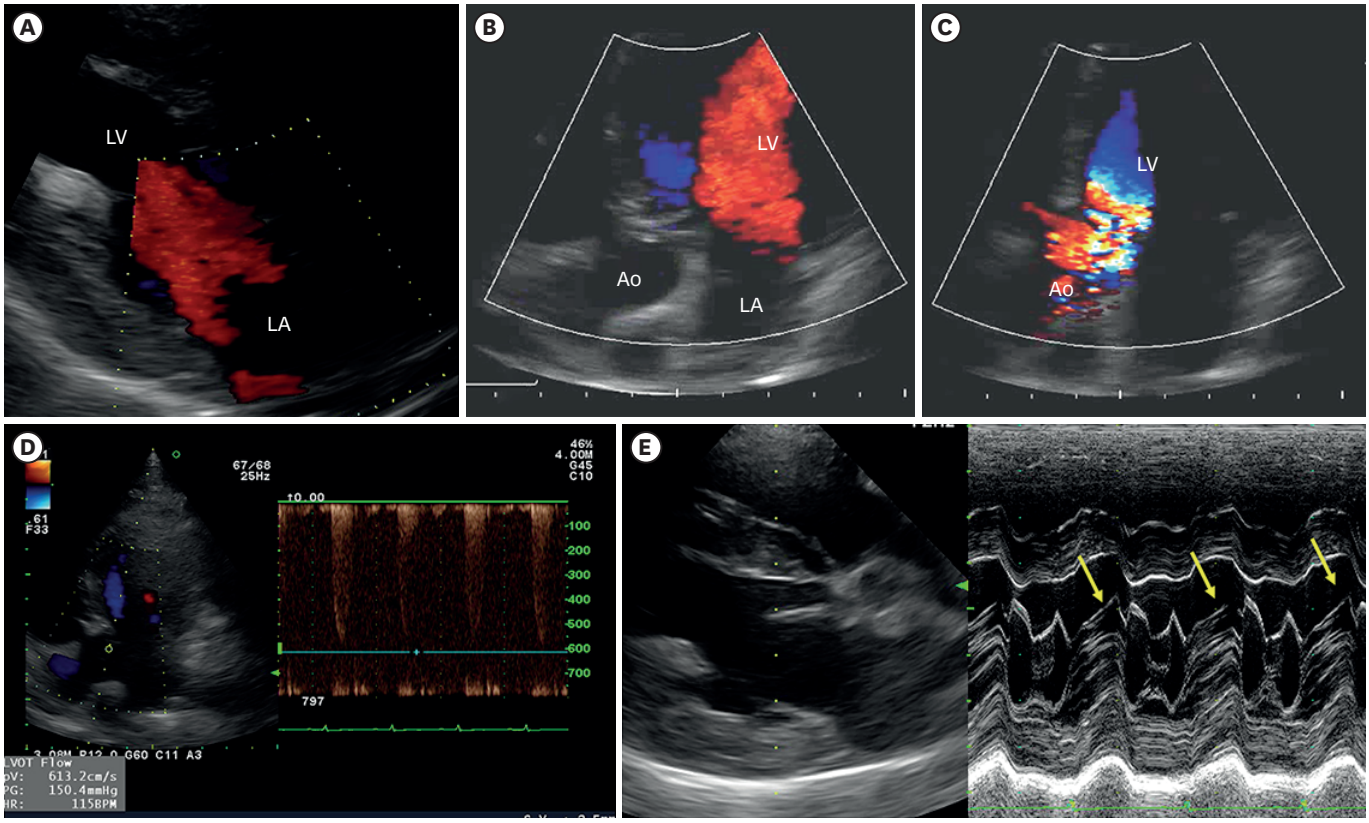

Fig. 3. Echocardiography of tilted right parasternal 4 chamber view (A), left apical LVOT view (B-D), and M-mode image at the level of the mitral valve of right parasternal four chamber view. (A, B) The patient had a normal mitral inflow during diastolic phase. (C) During systolic phase, there is no evidence of mitral regurgitation. Turbulent flow below the aorta and mild aortic insufficiency are observed. (D) The LVOT velocity is severely elevated (6.1 m/s) on spectral Doppler. (E) On M-mode image, the anterior leaflet showed systolic anterior motion (arrows) in mid-systole.
LVOT, left ventricular outflow tract.

Authors declare no off-label use of antimicrobials. The study was reviewed and approved by the hospital administration.

DISCUSSION

This report presented a dog with LVOT obstruction caused by AMVT and described the echocardiographic findings of AMVT. AMVT is a rare congenital cardiac anomaly, and based on our review of the literature, this is the first report of AMVT in dogs.

In humans, although various appearances including net-shaped, ridge, parachute-like shapes exist, AMVT usually has a leaflet shape with well-developed chordae and is attached to the anterior MV and its vicinity [1,2,4-6]. However, in this case, AMVT was connected with both the IVS and chordae tendineae, and the connected tendon was in turn associated with the PM and chordae tendineae of the anterior MV. Interestingly, AMVT in our case showed both mobile part connected to the chordae, and a fixed part attached to the ventricular septum, which was a unique feature compared to previously reported cases [2,7].

AMVT can induce LVOT obstruction via both the mass effect of AMVT itself and complications secondary to AMVT, such as fibrous deposition, thrombus, or remodeling

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of the cardiac muscle and, 86.6% of human patients with AMVT have LVOT obstruction [2,3,7,8]. The pressure gradient of LVOT in the previous cases varied from 10 to 130 mmHg; notably, a few patients had no gradient [2-4,9,10]. In general, when the pressure gradient increases over 50 mmHg, the patients become symptomatic [1,6]. Similarly, our case had LVOT obstruction with a relatively high pressure gradient (approximately 150 mmHg); The fixed part of the AMVT caused steady obstruction, and the mobile part accelerated the obstruction during the systolic phase by moving toward the LVOT.

The fixed part of the AMVT, in our case, appeared as a linear structure similar to a subaortic fibrotic band leading to subvalvular aortic stenosis. Typical subaortic stenosis related to a fibrous tissue could be ruled out in our case because the abnormal structure below the AV was not isolated but continuous to chordae tendineae of the MV [11]. In addition, LV false tendon and anomalous PM insertion could be differentiated from AMVT in our case. LV false tendons appear as linear structures between the LV wall and IVS, PM, or other part of the LV wall [11]. The connection of LV false tendon is found between normal structures but not with accessory tissue. The echocardiographic feature of anomalous PM insertion is a large muscle with short chordae extending from the anterior MV; however, our case had relatively long chordae and a well-developed PM [12]. The vegetation, redundant MV chordae tendineae, and cardiac tumors were also excluded from the differential diagnosis [6]. These conditions may be considered in other types of AMVT, such as net-shaped AMVT or nodular AMVT [3,5,13]. However, there was a marked difference in the appearance of these conditions from of our case.

Moreover, in our case, the mobile part of the tissues attached to the MV induced SAM of the anterior leaflet. The concurrence of SAM is not a frequent finding in AMVT in humans and has only been reported in a few patients with the mobile-type AMVT [2,14]. All of them had mild to severe MR generated by SAM of the anterior leaflet and incomplete coaptation of the MV. Interestingly, in our case, MR was not developed, although SAM of the anterior MV leaflet occurred consistently. SAM of the MV in the absence of MR occurs because proper coaptation of the MV is maintained when both leaflets are elongated or when the posterior MV leaflet with increased mobility moves toward the anterior leaflet [15]. There has been a report regarding a close inverse correlation between the contact length of the leaflets and MR, and an inverse correlation between MR jet area and post-MV mobility [15]. Since echocardiography showed that the posterior MV was slightly toward the anterior MV and IVS, we suspected that the absence of MR was due to the latter cause in our case.

AMVT may be isolated or accompanied by other cardiac defects, such as ventricular septal defect [2,3]. In human AMVT, two-third of patients had other congenital cardiac anomalies [2]. When comorbid conditions exist, symptoms are more likely to appear, and the age at the time of diagnosis is younger [2]. In our case, AMVT had developed without other cardiac anomalies. Thus, there may be no serious clinical signs even though the high pressure gradient of LVOT obstruction was present.

This case has several limitations. First, the presence of AMVT wasn't confirmed by surgery or necropsy. However, it is known that diagnosis can be made by differentiating the diseases that cause LVOT through echocardiography in human. Second, the clinical significance and prognosis of this case could not be determined because the follow up was lost. Third, the cause of the aortic insufficiency was not clearly determined in this case. From B-mode, aortic valve itself had no morphological deformities with normal movement and there was no evidence of relation of the aortic valve and AMVT, although there is a uncertainty due to lack

of necropsy. However, our case showed only mild aortic insufficiency and it was not severe enough to have any clinical significance.

In conclusion, this report presents the first case of AMVT with echocardiographic findings in a dog. Although many features of AMVT in dogs have not been characterized, echocardiography plays a pivotal role in the diagnostic process. AMVT can be an incidental finding in younger patients, even without other comorbid cardiac anomalies or distinct symptoms, however AMVT should be considered a rare but a possible cause of LVOT obstruction.

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