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Short Communication / *Kısa Bilimsel Çalışma*

Atrial septal aneurysm mimicking cor triatriatum in a Cavalier King Charles dog

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Summary: Atrial septal aneurysm is a rare deformity of interatrial septum extending to changeable directions during cardiac cycle. A 9 year old Cavalier King Charles Spaniel referred to Small Animal Hospital with a history of acute onset of respiratoric distress and abdominal distension. Routin blood work, electrocardiography and thorax radiography were performed. Echocardiography revealed mitral valve disease and severe left atrial dilatation with an atrial septal aneurysm as a bulging of interatrial septum into the right atrium and nearly prolapsing into the tricuspid orifice. Color flow Doppler revealed a left-to-right shunt flow atrial septal defect. The dog died suddenly 2 weeks later. The owner declined postmortem examination. To the best of authors' knowledge, this was the first report of atrial septal aneurysm with concomitant atrial septal defect in a dog. The purpose of this case report is to point out atrial septal aneurysm as the possible complication of mitral valve disease in a Cavalier King Charles Spaniel.

Keywords: Aneurysm, atrial septal defect, mitral valve disease.

Cavalier ırkı bir köpekte cor triatriatum'u taklit eden atrial septal anevrizma

Özet: Atrial septal anevrizma, interatrial septumun bir kalp siklusu süresince değişken yönlerde yönelimini ifade eden nadir bir deformitedir. 9 yaşlı, Cavalier ırkı köpek küçük hayvan hastanemize, akut olarak başlayan respiratorik güçlük ve abdominal genişleme şikayetleriyle getirildi. Hastaya rutin kan analizleri, elektrokardiyografi ve toraks radyografileri uygulandı. Ekokardiyografik muayenede mitral kapak hastalığı ile triküspid orifis ve sağ atrium içine doğru interatrial septum boyunca prolabe olmuş septal anevrizmanın eşlik ettiği şiddetli sol atrial dilatasyonu ortaya koydu. Renkli Doppler ile sol-sağ şant akımlı atrial septal defekt belirlendi. Hasta 2 hafta sonra aniden hayatını kaybetti. Hasta sahibi postmortem muayeneyi kabul etmedi. Yazarların bilgisine göre; bu olgu, bir köpekte atrial septal defektin eşlik ettiği ilk atrial septal anevrizma raporudur. Bu olgu ile Cavalier ırkı bir köpekte, mitral kapak hastalığının olası komplikasyonu olarak atrial septal anevrizmanın vurgulanması amaçlanmıştır.

Anahtar sözcükler: Anevrizma, atrial septal defekt, mitral kapak hastalığı.

Atrial septal aneurysm (ASA) is a rare saccular deformity of interatrial septum bulging into the right or left atrium or both (3). It has not been adequately identified in veterinary literatures. However, cases of ASA with variable morphology and classification have been reported in human literatures, especially at the level of the fossa ovalis (1,9,11,16). Most reports in human literatures revealed the association of ASA with congenital or acquired heart diseases such as atrial and ventricular septal defect, mitral prolapse, myxomatous valve disease, abnormal structure of the interatrial septum and, the changes in interatrial pressure gradient (1,10). The case presented here closely reflects a form of ASA (Type 1R) secondary to myxomatous mitral valve disease in humans (1,2,12,15). The case also revealed atrial septal aneurysm with concomitant atrial septal defect in a dog. Although cases of acquired atrial septal defects secondary to mitral valve disease have been reported in most veterinary literatures, none of them includes the cases of atrial septal aneurysm (13).

A 8 kg, 9-year old, intact male Cavalier King Charles Spaniel breed dog with a history of respiratory distress, exercise intolerance, orthopnea and coughing referred to Small Animal Hospital of Veterinary Faculty, Ankara University. The dog was not receiving any medication at the time of referral investigation. Physical examination revealed poor orodental health status, mucosal pallor and increased capillary refill time (>3s). The dog was tachycardic (heart rate= 163 bpm), tachypneic (respiratory rate = 38-50 breaths per minute) with increased respiratory effort, and normothermic (38.6^{o}C). Grade IV/VI left apical systolic heart murmurs were ausculted. Femoral arterial pulses were slightly weak. Bilateral crackles were also ausculted in the caudal

lung fields. A rapid heartworm antigen test was negative. Thoracic radiographs indicated severe generalized cardiomegaly with pulmonary edema. Abdominal ultrasound revealed a minimal amount of abdominal effusion and hepatomegaly with gallbladder wall thickening. A standard 6-lead electrocardiogram (Cardiofax ECG 6851K, Nihon Kohden Corporation, Tokyo, Japan) displayed left atrial enlargement (P mitrale, P duration > 0.04s) with tall R waves in lead II (1.9 mV). 2D echocardiography (Sdu 450 Ultrasound System, Shimadzu, Nakagyo, Japan) showed diffuse thickening of the mitral leaflets, mitral prolapse, left atrial dilatation and eccentric hypertrophy of the left ventricle (Table 1). Color flow Doppler (DC-6 Expert Ultrasound System, Mindray, Shenzhen, China) revealed severe mitral regurgitation and moderate tricuspid regurgitation with significant doppler-detected pressure gradients. (Table 1). Congestive heart failure secondary to myxomatous valve degeneration was diagnosed considering the clinical signs and diagnostic applications. The dog was placed into an oxygen cage and initiated the following medications: pimobendan (0.3 mg/kg PO q12h), furosemide (1 mg/kg IV q12h) and lisinopril (0.5 mg/kg PO q24h). The symptoms decreased within 2 weeks following the medication. However, the dog had a cardiac stability during 3 months.

Three months later the dog developed severe peritoneal effusion and acute onset of respiratory distress. The dog was tachycardic (heart rate = 192 bpm) and hypothermic (37,4^{o}C). Grade V/VI left systolic heart murmurs with palpable precordial thrill were ausculted. Femoral arterial pulses were weak. A complete blood count was within normal limits. A serum biochemistry panel showed mild increases in urea (60.7 mg/dl, reference interval 15-59.9 mg/dl), creatine kinase (218 IU/L, reference interval <200 IU/L) and serum sodium concentration (155.3 mmol/L, reference interval 140-154 mmol/L). Analysis of abdominal free fluid indicated transudate with total protein <2.5g/dl. Electrocardiographic examination confirmed fine Atrial fibrillation (AF) with a rapid ventricular response (heart rate = 270 bpm). Wide QRS complex tachycardia was also present in lead aVL (Figure 1). Repeated 2D echocardiography revealed severe left atrial dilatation with an ASA as a bulging of interatrial septum into right atrium and nearly prolapsing into the tricuspid orifice (Figure 2). However, color flow Doppler revealed the atrial septum extending into the right atrium and a left-to-right shuntflow atrial septal defect (ASD) 3.3 mm in diameter (Figure 3). The dog was treated with a total dose of 15 mg/kg furosemide (3 x 5 mg/kg IV q6h), spironolactone once (2 mg/kg PO), diltiazem (1 mg/kg PO q12h) and aspirin (20 mg/kg PO q12h) with a supplemental oxygen and heating system. Nevertheless, the dog died suddenly 2 weeks later. The owner declined postmortem examination.

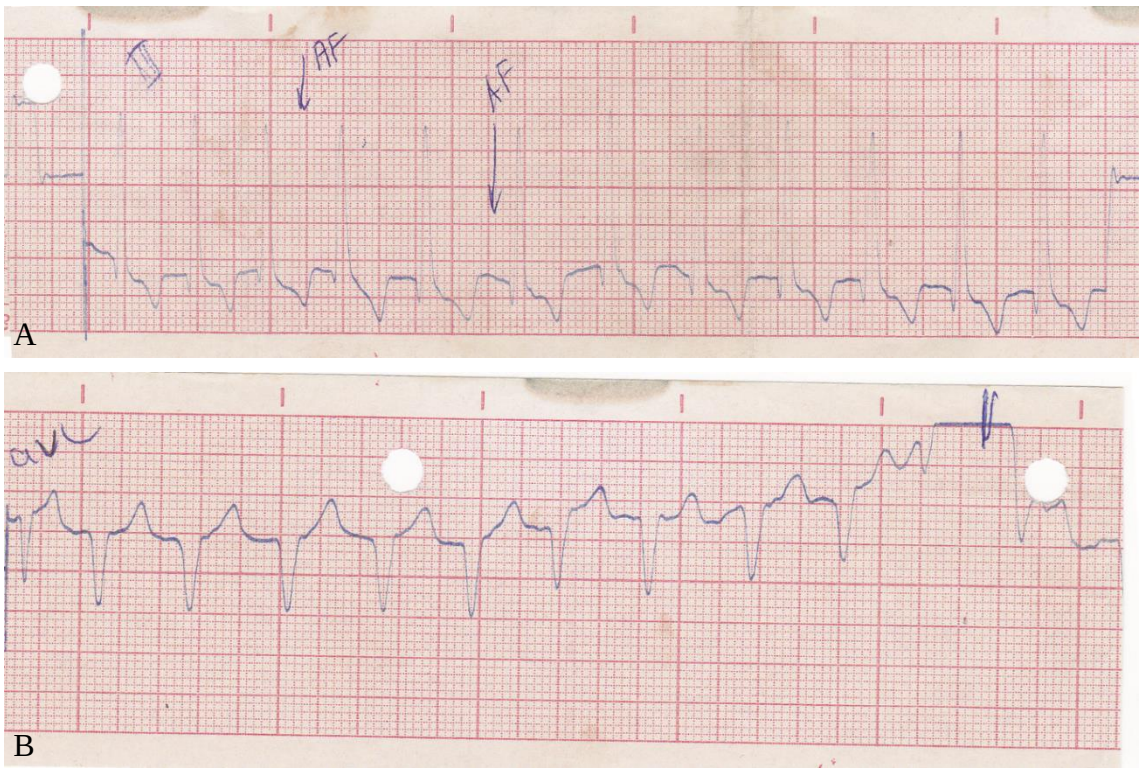
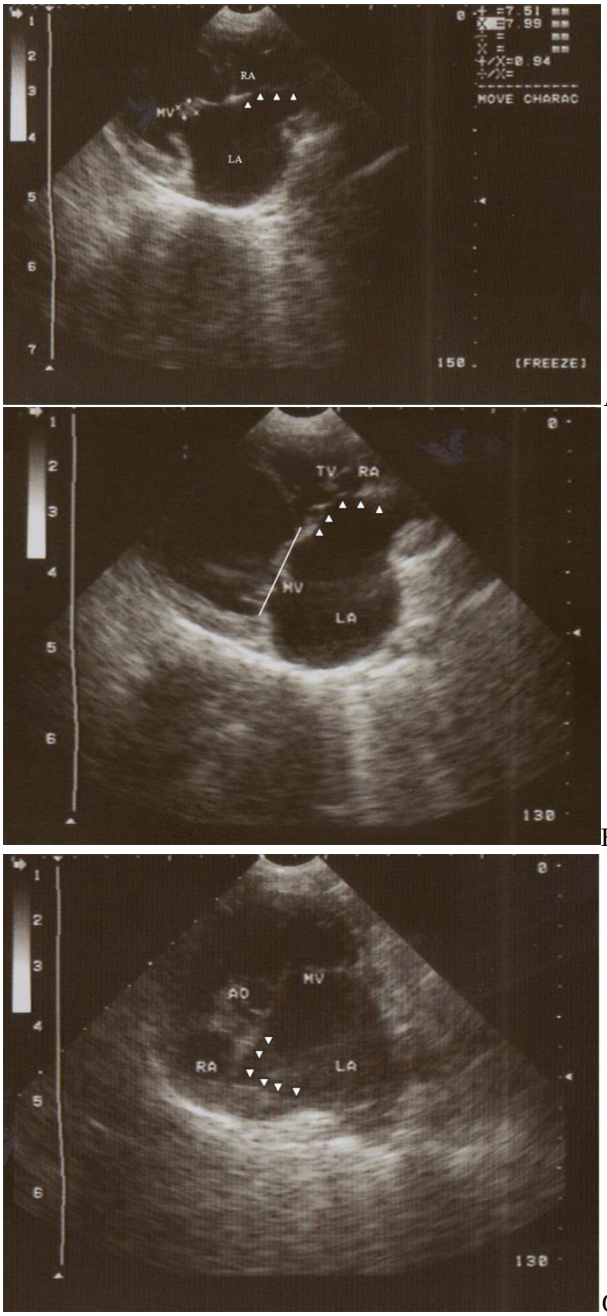


Figure 1. (A) Fine AF with an average ventricular response rate of 270 bpm and (B) wide QRS complex tachycardia, 0.04-0.06 ms (50 mm/s and 10 mm/mV, lead II and aVL, respectively).
Şekil 1. (A) Ortalama 270 atım/dk hızlı ventriküler yanıtlı ve ince dalgalı AF ve (B) geniş kompleks taşikardi, 0.04-0.06 ms (50 mm/s and 10 mm/mV, derivasyon II ve aVL).



A

B

C

Figure 2. (A) Rounding, curling and irregularity at the tips of the mitral valve leaflets in the dog. Severe left atrial enlargement (based upon a large left atrial to aortic ratio) and ASA as a bulging of interatrial septum into right atrium (arrowhead) is present (right parasternal long axis view). (B) Mitral valve prolapse and ASA nearly prolapsing into the tricuspid orifice (right parasternal long axis view). (C) Type 1R ASA protruded from the midline of the interatrial septum into the right atrium throughout the cardiac cycle (left apical five chamber view).

Şekil 2. (A) Hastanın mitral kapak uçlarında görülen yuvarlaklaşma ve kıvrılma. Şiddetli sol atrial genişleme (artmış sol atrium/aort oranı) ve interatrial septum boyunca sağ atrium içine doğru prolabe olmuş ASA (ok) dikkati çekmekte (sağ parasternal uzun eksen görünümü). (B) Mitral kapak prolapsı ve neredeyse triküspid orifis içine doğru yönelen prolabe ASA (sağ parasternal uzun eksen görünümü). (C) Kalp siklusu süresince, interatrial septumun orta hattından sağ atirum içine doğru prolabe olmuş Tip 1R ASA (sol apikal 5 boşluk görünümü).

Table 1. Echocardiographic measurements from the dog with severe mitral regurgitation and moderate tricuspid regurgitation.

Tablo 1. Mitral ve triküspid regürgitasyonlu hastadan elde edilen ekokardiyografik ölçümler.

Variables	Results	References[5]
IVSd (mm)	9	6.88-8.27
LVIDd (mm)	44	24.46-26.44
LVWd (mm)	7	5.5-6.64
IVSs (mm)	13	10.34-11.89
LVIDs (mm)	26.8	14.44-16.67
LVWs (mm)	10.6	9.11-10.53
LA (mm)	37.53	15.11-17.38
Ao (mm)	15.23	15.36-16.86
Higher MV E:A Ratio	Restrictive pattern	
MR Jet velocity	6.70 m/sec	
MR Max PG	179,56 mmHg	
Aortic flow velocity	1.45 m/sec	

IVSd, interventricular septum diameter during diastole; LVIDd, left ventricular internal dimension during diastole; LVWd, left ventricular wall diameter during diastole; IVSs, interventricular septum diameter during systole; LVIDs, left ventricular internal dimension during systole; LVWs, left ventricular wall diameter during systole (M-mode, 2D right parasternal short-axis view); LA, left atrial diameter; Ao, aortic diameter (2D right parasternal short-axis of the heart base); MV, mitral valve; MR, Mitral regurgitation.

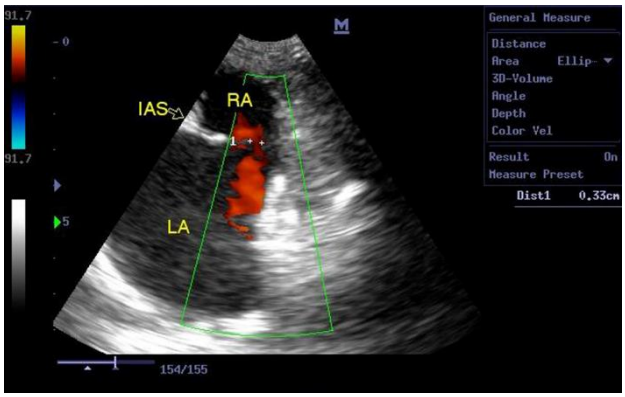


Figure 3. Color flow doppler shows the shunt flow through the defect from left to right.

Şekil 3. Defekt boyunca renkli akım doppleri ile sol-sağ şant akımının görünümü.

ASA with concomitant ASD in different interatrial pressures is an uncommon condition reported in humans (2,3). To the best of authors's knowledge, this was the first report of ASA with concomitant ASD in veterinary literatures. However, acquired ASDs secondary to mitral valve disease in two dogs have been described (13). Although echocardiograpy revealed a huge left atrium secondary to mitral regurgitation in the report, no dogs had ASA. Myxomatous mitral valve disease resulting in increased left atrial pressure and endocardial splitting in dogs has been identified (6). The extension of nonperforating splits over the fossa ovalis and additional mechanical stress on the atrial septum due to mitral

regurgitation or exaggerated motion contribute the atrial septal rupture (5). The case presented here were consistent with the reports previously described. The formation of ASA presented here is not only associated with severe mitral regurgitation and increased left atrial pressures, but also most likely associated with additional factors. The entity of interatrial pressure differences, abnormal flow patterns, primary malformation including the region of fossa ovalis and entire septum or the combination of these factors are the additional parameters in ASA formation (14). ASA has been reported in patients with normal atrial pressure as well (14). In the case presented here, although we did not obtain the pressure gradients among two atriums, flow across the atrial septal defect or Qp:Qs ratio, we suggest the combination of all these factors was the possible cause of ASA.

The effect of pimobendan in dogs with mitral regurgitation remains controversial. Some reports have suggested increased mitral regurgitant jet areas caused by long term pimobendan monotherapy in dogs with asymptomatic mitral valve disease (7,8). In addition, pimobendan increases mitral regurgitation and mitral valve lesions in dogs with mild degenerative mitral valve disease (7). However, short term pimobendan causes dose dependent decrease in left atrial pressure in dogs with mitral regurgitation (17). Although pimobendan administration was performed with lisinopril and furosemide in the present case with severe mitral regurgitation, we suggest that the another possible cause of ASA was the adverse effects of long term pimobendan on mitral valves, and therefore progressive left atrial pressure.

ASA has been classified in 5 types in human literatures (1). Protrusion of the interatrial septum from the midline into the right atrium throughout the cardiac cycle has been called type 1R ASA. The case presented here closely reflects a form of ASA (Type 1R) secondary to myxomatous mitral valve disease in humans (1, 12, 15).

ASA is a rare deformity of interatrial septum not adequately identified in veterinary literatures. This case report indicates ASA as the possible complication of mitral valve disease in a Cavalier King Charles Spaniel. Therefore, ASA should be considered as the possible complication of mitral valve disease and added in relevant sections of veterinary cardiology books.

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