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<Case Report>

Persistent left cranial vena cava with congenital heart defect in two dogs

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Abstract: The purpose of this report is to introduce persistent left cranial vena cava (PLCVC) with persistent right aortic arch (PRAA) and patent ductus arteriosus (PDA). Case 1 was a Cocker Spaniel with PRAA and case 2 was a Maltese with PDA. PLCVC was enclosed at the sites of PRAA and PDA surgery; therefore, it was lifted dorsally during PDA and PRAA surgery. Surgery to repair congenital heart defects including PRAA and PDA is recommended for dogs that do not die of PLCVC at a young age.

Keywords: congenital heart defect, patent ductus arteriosus, persistent left cranial vena cava, persistent right aortic arch, thoracotomy

During fetal life, dogs normally have bilateral symmetrical cranial and caudal cardinal veins. As fetus developed, the right cranial cardinal veins direct fused and transformed into right cranial vena cava [3, 5]. Left cranial cardinal veins usually become atrophied and left caudal cardinal veins develop into coronary sinus. But in persistent left cranial vena cava (PLCVC) there is abnormal development of the sinus venosus and left cranial cardinal veins are not atrophied and remain the left common cardinal vein at the coronary sinus [3]. There are two types of PLCVC reported in the dog, a complete one in which the non-atrophied left cranial cardinal vein retains its embryological connection with the coronary sinus [11] and an incomplete one where the distal portion of the left cardinal vein atrophies as normal, but the proximal portion of the left cranial vena cava receives a vein which drains the left costocervical vertebral trunk [5]. PLCVC is an uncommon vascular anomaly but coexists with other congenital heart defects [6, 12]. PLCVC itself did not show significant clinical signs in most cases but mostly detected when cardiovascular imaging performed for other reasons [1, 6]. A study in dogs reported that 12% of vascular rings was consisted of an association of persistent right aortic arch (PRAA) and PLCVC. In these cases clinical signs of esophageal disease due to PRAA are usually appear [2]. If PLCVC drains into right atrium, there is no clinical significance [17]. In human medicine PLCVC called persistent left superior vena cava (PLSVC). In numerous instances PLSVC was found accidentally during cardiovascular imaging or surgery

[9]. Although it is present up to 0.5% in general population without congenital abnormalities [10, 15]. Almost 40% of the patient show different kinds of congenital anomalies like as coronary sinus ostial atresia, atrial septal defect, coarctation of aorta, and bicuspid aortic valve [13, 15, 18]. It can cause arrhythmias and cardiogenic shock due to secondary causes such as physiologic stresses placed on the conductive tissue due to abnormal anatomy that can lead to the enlargement of the right atrium [14]. Many types of PLSVC are present in human medicine, hence, many types of treatments exist like PLSVC anastomosed to right atrial appendage, cardiac catheterization and pacemaker implant [8, 9, 16]. In veterinary medicine, as diagnostic tool development, PLCVC is diagnosed by angiocardiography and magnetic resonance imaging (MRI) [7], while electrocardiographic and echocardiographic findings give little information about PLCVC [5].

The purpose of this rare case report is to introduce PLCVC with PRAA and patent ductus arteriosus (PDA).

Data for 2 dogs with PLCVC were PRAA and PDA, respectively. First case, a 1-year-6-month-old female, 6.12 kg Cocker Spaniel was admitted to veterinary medical center with chronic vomiting. Clinical problem was onset 4 months ago. At the time of admittance hospital, body temperature and blood pressure were normal range, but respiratory sounds were honking sound and panting. After thoracic and abdominal radiography were conducted, radiography showed sign of mega-esophagus caused by obstruction. After barium contrast study was performed, obstruction site was clearly found

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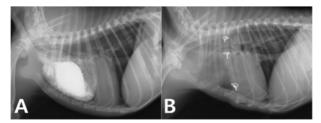


Fig. 1. (A) Thoracic radiography by barium contrast study shown enlargement of esophagus caused by obstruction. (B) Obstruction site is relieved and chest tube is replaced after surgery.

(Fig. 1A). Clinical signs and radiological findings presented patient was PRAA. Blood chemistry and CBC results showed patient body conditions in inflammation state because of aspiration pneumonia according to regurgitation. Patient nutrient state was also poor caused by chronic vomiting. Surgery was performed 20 days after admission in hospital with antibiotic therapy and nutrient state correction. During surgery PLCVC was detected in upper side of heart (Fig. 3A) and was carefully dissected and lifted dorsally. Perioesophageal fibrous tissue was dissected and then endo-esophageal tube was passed down into the obstruction site for confirming the opening. Chest tube was placed in thoracic cavity. After surgery, radiography showed obstruction site that was relieved and chest tube was found (Fig. 1B). Patient was managed to eat liquid food with upright posture during 3 weeks after surgery. Two months after surgery, patient had no cough and regurgitation, and body condition was good with gained weight.

Second case, a 2-month-old female, 1.18 kg Maltese was admitted to veterinary medical center with chest thrill. On physical examination, continuous murmur with chest thrill was detected. Other body conditions, blood pressure, body temperature and blood chemistry were normal. Radiological finding showed cardiomegaly, generalized dilation of pulmonary vessels, main pulmonary artery (MPA) bulging, and enlargement of left atrium (LA) and left ventricle (LV) (Fig. 2A). Furthermore, echocardiography finding presented abnormal systolic flow in the MPA, LV systolic dysfunction, showed shunting orifice, dilated LA/aorta ratio and enlargement of LA and LV. Also cardiac MRI image showed shunting orifice called PDA (Fig. 2C) and abnormal vessel called PLCVC (Fig. 2D). Due to patient's age, surgery was delayed 3 months. As thoracotomy was proceeded, PLCVC was detected and located in exactly upper side of the PDA (Fig. 3B). Thus PLCVC was carefully dissected and lifted dorsally to secure a clear PDA surgical view. Then PDA was ligated with black silk. Heart continuous murmur and chest thrill were disappeared right after surgery. Patient monitored for 8 months after surgery, was accompanied by radiography. The radiography showed that heart size was returned to normal condition (Fig. 2B).

The present case report describes the detection of PLCVC during congenital heart defects like PRAA and PDA in dogs. Two dogs were completely return to normal life and there were no complications related with heart disorders for more

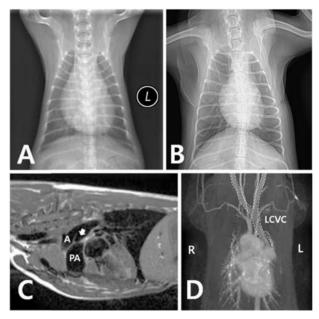


Fig. 2. (A) Radiological finding show cardiomegaly, main pulmonary artery (PA) bulging, enlargement of left artrium and left ventricle. (B) After 8 months, heart size is returned to normal condition. (C) Magnetic resonance imaging (MRI) shows patent ductus arteriosus (PDA) which located between aorta (A) and PA. (D) MRI identify abnormal vessel called persistent left cranial vena cava (PLCVC).

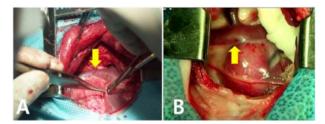


Fig. 3. PLCVC is shown during persistent right aortic arch (A) and PDA (B) thoracotomy. Arrow indicates PLCVC.

than 2 years follow-up. During the normal process of canine embryo development, the venous return to the heart is through the paired cranial and caudal cardinal veins that join to form the common cardinal veins. The right and left common cardinal vein empties into the transversely positioned sinus venosus that develops into the right atrium. The right and left cranial cardinal veins are joined, later by direct fusion cranial to the heart. The remainder of the left cranial cardinal vein atrophies caudal to the fusion, while right side vessel enlarged and became the distal part of the cranial vena cava. The proximal part of the right cranial vena cava is formed by the right common cardinal vein and most part of left cardinal system become atrophied, only the left common cardinal vein persists, forming the coronary sinus. Myocardial venous blood drains into the coronary sinus and then into the right atrium [10].

In dogs, PLCVC is relatively rare. However, it is generally accompanied with congenital heart disease, rather than present only itself. PLCVC could be identified during surgical correction of congenital heart disease. Most of dogs with PLCVC and other congenital heart problems can cause sudden death in young ages. Although dogs with PLCVC are survived, other congenital heart defect is still a threat to lives and causes euthanasia in several veterinary cases [7, 12].

In present case report during the surgery of PRAA patient PLCVC was detected in upper side of heart. In PDA surgery case, as the thoracotomy was proceeded, PLCVC was detected and located in exactly upper side of PDA. PRAA denotes the vascular ring anomalies associated with secondary megaesophagus [4]. A distended esophagus is generally recognized as PRAA. In the present case there was incomplete atrophy of the cranial cardinal vein enclosing esophagus with the trachea and causing constriction of the esophagus.

In conclusion, this case report PLCVC was described with combination defect of PRAA and PDA in dogs. Dogs which do not expired by PLCVC in young age are encouraged to have surgery of congenital heart defect including PRAA and PDA that can be directly related to life threatening condition. Thereby it increases the survival rate of PLCVC patients with congenital heart defects.

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