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Epidemiological and Morphological Studies of Double-Chambered Right Ventricle in Dogs

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ABSTRACT. The double-chambered right ventricle (DCRV) is a rare congenital cardiac disease in dogs, and its detailed epidemiological and morphological features are not clearly understood. By investigating the profile, clinical signs, and characteristics of examination findings of eleven dogs with DCRV by means of a retrospective study, we attempted to clarify the epidemiology and morphology of the condition. The study group consisted of nine males and two females. Breeds included Pug (n=3), Miniature Dachshund (n=1), French Bull-dog (n=1), Shiba (n=1), and Retrievers (n=5). The attachment site of the anomalous muscular bundle was continuous with the cardiac apex in nine dogs, and it was attached to the right ventricle free wall in the other two dogs. In dogs with DCRV, at least one of the following conditions was present concurrently: congenital or acquired tricuspid valve regurgitation (TR), ventricular septal defect, and atrial septal defect. Also, the pressure difference between the two chambers increased over time, and progressive right-sided heart failure was observed. In summary, DCRV occurs in small breeds of dog as well as in large breeds of dog and it may be more prevalent in males. The existence of two types of DCRV in dogs was established. Dog with DCRVs will have a high incidence of concurrent cardiac abnormalities. Concurrent TR may be either congenital or acquired. DCRV is a congenital disorder, but the clinical condition progresses as the dog develops.

KEY WORDS: canine, diagnosis, double-chambered right ventricle, echocardiography, epidemiological research.

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The double-chambered right ventricle (DCRV) is a congenital cardiac anomaly, first reported in human medicine in 1858 [15]. Because there are few reports of DCRV in dogs [14, 16, 22], its detailed epidemiological and morphological features are not clearly understood. In DCRV, the right ventricle (RV) is divided by an anomalous muscular bundle and a muscular membranous structure to form a proximal high-pressure chamber that includes the inflow portion, and a distal low-pressure chamber that includes the infundibular portion [12]. The high-pressure chamber and the low-pressure chamber communicate with each other only through several ostia, resulting in the generation of a marked pressure gradient between the two chambers [12]. Therefore, dogs with DCRV develop severe right-sided heart failure as the clinical state progresses [12]. Until now, this was all the information that was available on DCRV in dogs [14, 16, 22]. By investigating the profiles, clinical signs, and characteristics of examination findings for eleven dogs with DCRV, we attempted to clarify the epidemiology and morphology of DCRV in dogs.

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MATERIALS AND METHODS

Subject profile: The style of this research is that of a retrospective study. Between June 2001 and December 2010, one dog was diagnosed with DCRV at the Animal Clinical Research Foundation (Tottori), four dogs were diagnosed at the Tokyo University of Agriculture and Technology Animal Medical Center (Tokyo), and six dogs were diagnosed at the Nippon Veterinary and Life Science University Veterinary Teaching Hospital (Tokyo). The body weights of these dogs ranged from 3.1 to 31.5 kg (median 9.2 kg), and their ages at initial presentation ranged from 5 to 66 months (median 18 months). There were nine males and two females. Breeds included Pug (n=3), Miniature Dachshund (n=1), French Bull-dog (n=1), Shiba (n=1), Labrador Retriever (n=2), Golden Retriever (n=2), and Flat-coated Retriever (n=1) (Table 1).

Examination: Information on the clinical history and clinical signs was obtained, and physical examination, thoracic radiography, electrocardiography, echocardiography, and blood examination were performed for all eleven dogs. Cardioangiography through the right jugular vein was performed in seven of the dogs. In all the dogs, thoracic radiographs were obtained from the right-recumbent position, and the lung field and the morphology of the heart were examined. In addition, the size of the heart was evaluated by using the vertebral heart score (VHS) [17]. Electrocar-

Table 1. Profile of clinical case

Dog	Age (months)	Body weight (kg)	Sex	Breed
1	5	4.3	Male	Pug
2	6	3.2	Female	Miniature Dachshund
3	6	6.7	Female	Shiba
4	12	24.3	Male	Golden Retriever
5	17	25.6	Male	Labrador Retriever
6	18	8.7	Male	Pug
7	19	31.7	Male	Labrador Retriever
8	22	23.5	Male	Golden Retriever
9	27	9.0	Male	Pug
10	36	25.5	Male	Flat-coted Retriever
11	66	9.2	Male	French Bull-dog

Profile taken at the initial presentation.

diography was performed by using the limb lead, with all the dogs in the right-recumbent positions. Echocardiography was performed in the right- and left-recumbent positions. The values from reports by Goncalves *et al.* [9] were used as reference ranges for the thickness of the interventricular septum (IVS) and of the left ventricle (LV) free wall. Also, the normal thickness of the RV free wall was considered to be half that of the LV free wall [2]. Therefore, when the thickness of the RV free wall was over half that of the LV free wall, this was taken as representing hypertrophy of the RV [2].

For the blood examination, complete blood cell counts were measured and the concentrations of blood urea nitrogen, creatinine, alanine aminotransferase, total protein, albumin, creatine kinase, sodium, potassium, and chlorine in the serum were determined. For the cardioangiography, an 80-cm-long catheter with an external diameter of 4 Fr (1.35 mm) of the multipurpose Cournand type (TCA7118015; Technowood, Tokyo, Japan) was used. Atropine sulfate (Atropine Sulfate Injection 0.5 mg; Mitsubishi Tanabe Pharma Corporation, Osaka, Japan) was given subcutaneously at a dosage of 0.04–005 mg/kg as premedication in all dogs subjected to cardioangiography, and these dogs were induced using Propofol (Rapinovet, Intervet K. K., Tokyo, Japan) at a dosage of 4–6 mg/kg given intravenously 10 min after the atropine. Tracheal intubation was then performed under maintenance anesthesia with isoflurane (Isoful; Dainippon Sumitomo Pharma Co., Ltd., Tokyo, Japan); positive-pressure ventilation was used. In all cases, a contrast medium with an iodine content of 400 mg/kg (Oyparomin; Fuji Pharma Co., Ltd., Tokyo,) was used. All examinations other than cardioangiography were performed without sedation or anesthesia in all dogs.

RESULTS

History: Cardiac murmur was detected in all dogs at the time of their first or second vaccination. All dogs showed one or more of the following clinical signs: cough, exercise intolerance, cyanosis, panting, and syncope. Veterinarians referred all the dogs to our referral facilities for further investigations of their cardiovascular systems.

Clinical sings: Coughs were noted in five of the dogs (Dogs. 2, 3, 5, 6, and 11), exercise intolerance in nine dogs (Dogs. 1–5, 7, and 9–11), cyanosis in six dogs (Dogs. 3, 5, 7, 8, 10, and 11), panting in eight dogs (Dogs. 1–3, 5–7, 10, and 11), and syncope in six dogs (Dogs. 1, 5, 7, and 9–11). Nine dogs exhibited more than two of these clinical signs (Dogs. 1–3, 5–7, and 9–11). The clinical signs were noticed by the owners when the dogs were between 1 and 27 months of age (median 10 months).

Physical Examination: Body temperature was within the normal range in all dogs. The visible mucous membranes were pale in six of the dogs (Dogs. 3, 5, 7, 8, 10, and 11) and normal in the others. On thoracic auscultation, Grade III/VI systolic ejection murmur was detected in four dogs (Dogs. 1, 6, 8, and 10), Grade IV/VI murmur was found in six dogs (Dogs. 2–5, 7, and 9), and Grade V/VI murmur was found in one dog (Dog. 11). The points of maximum intensity of these murmurs varied and included the left heart apex in one dog (Dog. 1), the left heart base region in three dogs (Dogs. 6, 8, and 11), the right heart base region in four dogs (Dogs. 2, 4, 5, and 10), and both sides of the heart base in three dogs (Dogs. 3, 7, and 9). However, the pulmonary sound on auscultation did not reveal any abnormalities. Distension of the jugular vein was noticed in four dogs (Dogs. 5–7 and 10).

Thoracic Radiographs: The range of VHS values was 9.9 to 14.2 with a median of 10.9 (the normal range 8.6–10.5 vertebral units) [17]. Cardiac enlargement was present in eight dogs (Dogs. 3–8, 10, and 11), and this was accompanied by left-sided cardiac enlargement in five dogs (Dogs. 4, 5, 7, 8, and 10). One dog showed only left-sided cardiac enlargement with no right-sided enlargement (Dog. 6), and three dogs did not show any abnormalities in their cardiac silhouettes (Dogs. 1, 2, and 9). No enlargement of pulmonary artery was recognized in any of the dogs. Pleural effusion was confirmed in three dogs (Dogs. 5, 7, and 10).

Electrocardiography: All the dogs showed sinus rhythms and their heart rates were within the normal range [21]. Left-band branch block was observed in one dog (Dog. 5). A pulmonary P wave was confirmed on Lead II in five dogs (Dogs. 4–6, 8, and 10: range 0.45–0.80 mV, median 0.50 mV; normal range <0.4 mV) [21]. A right-axis deviation was found in five dogs (Dogs. 4, 6, 7, 9, and 11: range +112°

to +157°, median +140°), and a left-axis deviation was found in one dog (Dog. 10: +30°). The normal range of mean electrical axis is +40° to +100° [21].

Echocardiography: Two-dimensional echocardiography showed marked hypertrophy of the interventricular septum (IVS) and the RV free wall on the right parasternal long-axis four-chamber and the left ventricular outflow tract views in all the dogs. The IVS measured 1.5–6.6 mm more than the normal range, and the RV free wall measured 2.3–6.5 mm more than the normal range. On the other hand, no hypertrophy of the LV free wall was observed. From the same views, an anomalous muscular bundle extending from the cardiac apex or from the RV free wall near the cardiac apex to the infundibular portion was observed. The site of attachment of the anomalous muscular bundle was continuous with the cardiac apex in nine of the eleven dogs (Dogs. 1, 2, 4, 6, and 8–11) and it was attached to the RV free wall in the remaining two dogs (Dogs 3 and 7). In the right parasternal short-axis view at the level of the aortic valve, the distance between the attachment site of the anomalous muscular bundle and the pulmonary valve was closer in two dogs (Dogs. 3 and 7) than in the other nine dogs. The RV was divided into two compartments by an anomalous muscular bundle. The anomalous muscular bundle could also be clearly seen in the right parasternal short-axis view at the level of the tendinous cord. On the same view, hypertrophy of the IVS and the RV free wall was observed, accompanied by flattening of the IVS on the RV inflow tract side (Fig. 1). By means of color-flow Doppler analysis, turbulent flow was identified in the region of the anomalous muscular bundle. Continuous-wave Doppler analysis was used to measure the blood flow from the proximal chamber to the distal chamber and this was found to in the range 2.7 to 6.3 m/sec (median 4.6 m/sec). On the basis of this measured blood flow value, the pressure difference between the distal chamber and the proximal chamber was calculated by using the simplified Bernoulli equation [pressure difference=4 × (velocity^2)] to be between 28.7 and 156.3 mmHg [7] (Fig. 2).

Marked right-atrial enlargement was seen in seven dogs on the left and right parasternal, long-axis, four-chamber views. Additionally, the presence of tricuspid valve regurgitation (TR) was confirmed in ten dogs from the color-flow Doppler analysis. The speed of regurgitation at the tricuspid valve was measured to be 2.6 to 4.8 m/sec (median 3.8 m/sec) by the continuous-wave Doppler analysis. Among the ten dogs with TR, reduced opening and closing movements of the tricuspid valve and a short tendinous cord attached to the septal leaflet of the tricuspid valve were observed in four dogs (dogs 4, 6, 9, and 10) (Fig. 3). Additionally, the presence of a Kirklin type II ventricular septal defect (VSD) was confirmed in two dogs (Dogs 2 and 3) [6]. Among the dogs with VSD, the presence of right-to-left shunting blood flow through the defect was confirmed in one dog (Dog 3) by means of the color-flow Doppler analysis. Furthermore, this dog also had a concurrent patent foramen ovale-type atrial septal defect (ASD) [10]. Since the two dogs with VSDs did not possess an overriding aorta, the presence of a tetralogy

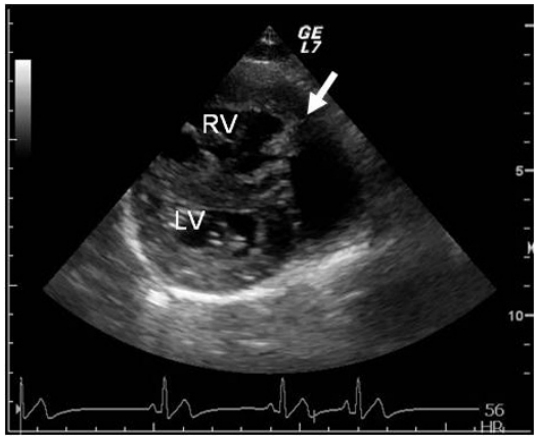


Fig. 1. Two-dimensional echocardiographic findings for the right parasternal short-axis view. The presence of an aberrant muscle band at the subinfundibular portion was confirmed (arrow). Flattening of the interventricular septum at the right-ventricular inflow tract side was also observed.

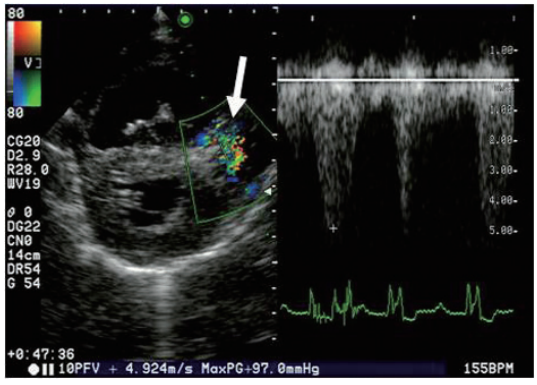


Fig. 2. Color-flow Doppler and continuous-wave Doppler echocardiography findings for the right parasternal short-axis view at the mitral valve level. With color Doppler flow imaging, turbulent flow was found to occur in region of the aberrant muscle band. With continuous wave Doppler, a high-velocity flow of 4.9 m/sec from the proximal chamber of the right-ventricular inflow tract toward the distal chamber at the infundibular was detected. The pressure difference between the distal chamber and the proximal chamber in the right ventricle, calculated by using a simplified Bernoulli equation, was 97 mmHg.

of Fallot was ruled out. The blood-flow velocity at the aorta was normal in all the dogs. For the remaining dog, a high blood velocity of 2.3 m/sec was measured at the main pulmonary artery apart from the anomalous muscular bundle (Dog 6). Pulmonary valve stenosis was diagnosed in this particular case from the abnormal morphology and movement of the pulmonary valve. Pulmonary regurgitation was not identified in any of the dogs. No signs were found of patent ductus arteriosus (PDA) or any type of aortic steno-

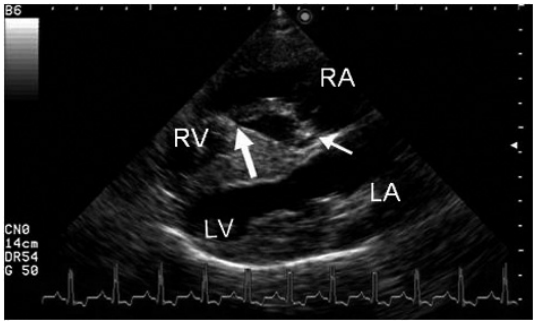


Fig. 3. Two-dimensional echocardiography findings for the right parasternal long-axis four-chamber view. The development of an aberrant muscle band from the lower third of the right ventricular wall toward the infundibular portion wall was confirmed (large arrow). Also, a thickening of the tricuspid valve septal leaflet, a short chordae tendineae which is attached to the septal leaflet, and a reduction in the opening and closing movements of the tricuspid valve were observed (small arrow).

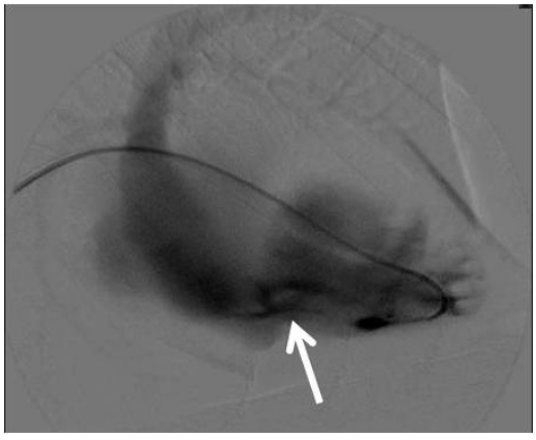


Fig. 4. Angiocardiography of a double-chambered right ventricle for the lateral view. The tip of the catheter can be observed within the right ventricle. The presence of an obstruction dividing the right ventricle into two chambers is confirmed (arrow).

sis. In this study, it was possible to perform Doppler analyses with a correction angle of less than 25° in all cases.

Blood examination: Polycythemia with a packed cell volume of 60% was observed in one dog with Kirklin type II VSD and ASD (Dog 3). The complete blood count and biochemical analyses for the remaining ten dogs showed no abnormalities.

Cardioangiography: The catheter was inserted from the jugular vein towards the apex of the RV. The systolic RV pressure of the area was measured by cardioangiography (35–127 mmHg; median 55 mmHg). Contrast medium was then injected from the tip of the catheter. Injection of the contrast medium revealed TR in six of the seven dogs subjected to cardioangiography. Additionally, a severe occlusion site was found at the mid-RV in four dogs (Fig. 4). However, because of the effect of the TR, confirmation of the occlusion site by use of the contrast medium was not possible in three dogs. Also, the presence of a right-to-left shunt was confirmed in the dog that was diagnosed with Kirklin type II VSD by echocardiography (Dog 3). Attempts were made to orient the catheter from the RV in the direction of the pulmonary artery in the seven dogs subjected to angiography. In four dogs, however, it was not possible to pass the catheter through the occlusion site within the RV, and it could not be advanced toward the pulmonary artery. In these cases, it was not possible to measure the pressure difference at the occlusion site. The pressure differences for the three dogs in which the catheter was able to pass beyond the occlusion site were 78, 100, and 121 mmHg, respectively (at ventricular systole).

Clinical outcome: All the dogs began receiving medical treatment at the latest after visiting our facility. Angiotensin-converting enzyme inhibitor was prescribed to all the dogs. Additionally, a beta-adrenergic blocking agent (either carvedilol at 0.1–0.4 mg/kg BID or metoprolol tartrate at 0.25 mg/kg BID) was prescribed to five dogs (Dogs 1, 5, 8, 9, and 11). Additional medications prescribed were as follows: furosemide (1–4 mg/kg BID) was prescribed in three dogs (Dogs 5, 6, and 8), isosorbide dinitrate (1–2 mg/kg BID) in five dogs (Dogs 1, 2, 5, 6, and 11), digitalis glycoside (0.005–0.01 mg/kg BID) in two dogs (Dogs 5 and 11), and dipyridamole (5 mg/kg BID) in two dogs (Dogs 3 and 8). At the time of the initial consultations, eight dogs were already receiving more than two kinds of medication. Later on, the drug dosage and the combination of the drugs were selected on the basis of the progression of the clinical state of the cardiac failure. As a result, the administration of more than three kinds of medication was needed in all the dogs.

In combination with the medical treatment, resection of the abnormal structure, which consisted of an anomalous muscular bundle and a muscular membranous structure, was attempted under the cardiopulmonary bypass in five of the eleven dogs (Dogs 3, 4, 7, 10, and 11). The surgical method of approaching the anomalous muscular bundle by incision into the RV was chosen in all the dogs. Echocardiography performed before the operation revealed that the pressure difference across the stenosis was in the range 78–155 mmHg, with a median of 101 mm Hg. In contrast, echocardiography performed just after the operation revealed the pressure difference across the stenosis to be in the range 4–41 mm Hg with a median of 25 mm Hg. The average rate of reduction of the pressure difference was 76%. In one of the five dogs that received surgery, paroxysmal ventricular tachycardia followed by ventricular fibrillation developed 30 hr after surgery. The dog was unresponsive to antiarrhythmic treatments and later died (Dog 4). Another dog died suddenly and unexpectedly one month after the surgery (Dog 11). The remaining three dogs were available for follow-up investigations. Echocardiography performed 10 months after surgery revealed the blood-pressure difference in the RV to be maintained in the range 30–50 mmHg. The

survival time of these three dogs after surgery was more than 50 months. However, the signs of right-sided cardiac failure gradually progressed in all three dogs. One dog later died at 91 months (Dog 7), but the remaining two dogs (Dogs 3 and 10) remain alive at the time of writing (ages 57 and 78 months, respectively).

Six of the eleven dogs received only medical treatment, with no surgical intervention (Dogs 1, 2, 5, 6, 8, and 9). Echocardiography was performed on these cases at the initial examination, and the range of pressure difference across the stenosis was 29–107 mmHg, with a median of 65 mm Hg. However, six months after the initial examination, the pressure difference across the stenosis had increased to 64–125 mmHg, with a median of 85 mmHg. Additionally, worsening of the TR was confirmed in these six dogs. Gradual worsening of the clinical signs related to the right-sided heart failure (including increased frequency of syncope, development of ascites, and pleural effusion) were seen in four of the six dogs and led to their deaths. One dog died suddenly 12 months after the initial consultation (Dog 9). The age at death of five of the six dogs was in the range 28–41 months, with a median of 30 months. The remaining one dog, aged 84 months, is still alive at the time of writing (Dog 8). In this case, the pressure difference across the stenosis at the initial examination was 28 mmHg, and this increased to 42 mmHg 24 months after the initial examination. Moreover, the frequency of syncope on excitement had increased, and it was determined that the clinical condition had been gradually progressing (Table 2).

DISCUSSION

The hemodynamics of DCRV is influenced by the pressure overload in the RV, and clinical progression leads to severe right-sided heart failure that is similar to pulmonary stenosis (PS) [16]. To date, many cases of PS in dogs have been reported, and the existence of breed predilection and gender difference for the various types of PS has been evident [4, 11]. This epidemiological information is frequently beneficial at the time of diagnosis. On the other hand, because there are few reported cases of DCRV, insufficient epidemiological information is available.

Willard *et al.* have reported cases in a boxer and in a Bouvier des Flandres [22]. Martin *et al.* have reported that five of the seven dogs exhibiting DCRV were either Boxers or Golden Retrievers [16]. For this reason, large breeds of dog were thought to be predisposed to DCRV [16]. Koie *et al.*, however, reported a case of DCRV in a Pug [14]. In our study, five of the eleven dogs were Retrievers, a similar proportion to that previously reported [16, 22]. However, the remaining six dogs were examples of small-to-medium sized breeds, of which three were Pugs. It is therefore apparent that DCRV in dogs is not limited to large breeds of dog, but also presents in small breeds of dog, such as Pugs. To date, DCRV has been reported to occur in dogs [14, 16, 22], cats [3, 13], and humans [1, 5, 8, 12, 13, 15, 18–20]. However, there have been no reports on association between the incidence of DCRV and gender predilection on any of these species. In the previous reports, two of the three dogs were males [14, 22]. In this study, nine of the eleven dogs were males. These findings suggest the existence of a gender predilection and the possibility of a higher incidence of the disease in male dogs.

In humans, DCRV is classified into two types, depending on the location of the anomalous muscular bundle. In the low oblique obstruction type, the attachment site of the anomalous muscular bundle is continuous with the cardiac apex, whereas in the high horizontal obstruction type, the anomalous muscular bundle is attached to the RV free wall [1, 5]. In our study, the presence of an anomalous muscular bundle that connects to the cardiac apex as well as one that connects to the RV free wall were confirmed by echocardiography. These findings confirmed the existence in dogs of the two types of DCRV (the low oblique obstruction type and the high horizontal type) that are also found in humans.

It has been reported that more than 60% of the cases of DCRV in humans are complicated by VSD [8]. Furthermore, aortic stenosis and PDA are known to occur concurrently [8]. However, in previous reports of DCRV in dogs [14, 16, 22], only one pug had been described as having con-

Table 2. Prognosis of dogs

Dog	Medication[a)]	Age at death (month old)	Surgery: age at the operation	Another abnormality[b)]	Typed of DCRV
1	A, B, I	28			Low oblique type
2	A, I	34		VSD	Low oblique type
3	A, Di	Alive: 57	Yes: 3	VSD + ASD	High horizontal type
4	A	41	Yes: 41		Low oblique type
5	A, B, D, F, I	30			Low oblique type
6	A, F, I	30		PS	Low oblique type
7	A	110	Yes: 19		High horizontal type
8	A, B, F, Di	Alive: 84			Low oblique type
9	A, B	41			Low oblique type
10	A	Alive: 78	Yes: 38		Low oblique type
11	A, B,D, I	68	Yes: 67		Low oblique type

a): medication at the initial presentation. A: angiotensin-converting enzyme inhibitor, B: be-ta blocker, D: digoxin, Di: dipyridamole, F: furosemide, I: isosorbide dinitrate. b): exclude tricuspid valve regurgitation. VSD: ventricular septal defect, ASD: atrial septal defect, PS:pulmonary valve stenosis.

current VSD and DCRV [14]. Many dogs with DCRVs in our study had concurrent cardiac abnormalities, such as VSD, ASD, PS, or tricuspid valve dysplasia. Therefore, it is likely that dogs with DCRVs will have a high incidence of concurrent cardiac abnormalities. In this study, the one dog with concurrent VSD in addition to high horizontal obstruction type of DCRV, a right-to-left shut at the deficit was observed. We hypothesize that the reason for this hemodynamic state is that the presence of VSD at the high-pressure chamber side causes the pressure of the high-pressure chamber to be above that of the LV. Furthermore, this dog differed from the other ten dogs, in that it showed clinical signs strongly suggestive of cyanotic heart disease, such as cyanosis or polycythemia. Therefore, when DCRV occurs in conjunction with another concurrent cardiac anomaly, it is possible that various hemodynamic states and clinical signs might be displayed.

In dogs, the rate of concurrence of TR with DCRV is high; Martin *et al.* reported this rate to be 57% (four of the seven dogs) [16]. Of these four dogs, three showed restricted movement of the tricuspid valve leaflets on echocardiography, and tricuspid valve dysplasia was suspected [16]. In this study, ten of the eleven dogs showed mild-to-severe TR, and of these dogs, four showed abnormal morphology of the tricuspid valve leaflets and movements on echocardiography. Thus these four dogs were diagnosed as tricuspid valve dysplasia. Visual inspections of the tricuspid valves were possible on three of the four dogs, and these inspections confirmed the diagnosis of tricuspid valve dysplasia. On the other hand, the remaining six of the ten dogs were not diagnosed as tricuspid valve dysplasia by echocardiography. Of these dogs, visual inspections was possible in two dogs, we were able to confirm the absence of abnormal morphology of the tricuspid valves. It is therefore possible that TR can develop as both a congenital and an acquired condition. Additionally, because the results of echocardiography and visual inspections were in agreement, echocardiographic diagnosis of tricuspid valve dysplasia is thought to be possible and adequate.

In this study, varied results of blood examination, auscultation, electrocardiography, and thoracic radiography were obtained from the dogs. The reasons for the variations in results appear to be related to the severity of TR, coexisting cardiac abnormalities, and differences in the thoracic form. Therefore, we suggest that DCRV cannot be adequately diagnosed on the basis of these examinations alone.

Diagnosis of DCRV can be confirmed by the presence of an anomalous muscular bundle within the RV causing widening of the pressure difference within the ventricles at proximal and distal points of the anomalous muscular bundle. In this study, we identified the following characteristics in echocardiography that are peculiar to DCRV in all dogs; (1) visualization of an aberrant muscle band in the RV below the infundibular region, (2) detection of turbulent blood flow and excessive acceleration of the blood flow in the region of the anomalous muscular bundle, (3) flattening of the IVS in the RV inflow tract side, and (4) increased thickness of the proximal RV wall and papillary muscles [19]. These results confirm that echocardiography is a useful technique for diagnosing DCRV in dogs, in agreement with the results of Martin *et al.* [16].

In this study, we performed cardiac catheterization on seven dogs, but we were unable to obtain any additional useful information to that given by echocardiography. One possible reason is the presence of the anomalous muscular bundle and its continuing membranous structure, which may have blocked the passage of the catheters.

We also considered combined medical and surgical treatments or medical treatments alone as therapeutic options for dogs with DCRV. Survival times appeared to be longer for combined medical and surgical treatments; however, because of the small number of cases, this could not be proved. It was also not possible to show any differences in prognosis in relation to the types of medications given or the combinations of medications used. We hope to resolve this problem in the future as the number of cases of dogs with DCRV increases.

The occlusion site of the DCRV is formed by an anomalous muscular bundle and a muscular membranous structure that is continuous with the muscular bundle. For this reason, the high-pressure chamber and the low-pressure chamber communicate with each other through the ostium. We found that the pressure difference between the two chambers increased with age in dogs with DCRV. Worsening of the TR over time was also observed. Alongside these changes in hemodynamic status, clinical signs, such as pleural effusion and syncope, also became prominent. We hypothesize that this is probably due to hypertrophy of the anomalous muscular bundle or to development of the muscular membranous structure thereby causing a gradual narrowing of the ostium between the high-pressure chamber and the low-pressure chamber. Therefore, although DCRV is a congenital cardiac disorder, it is possible for the clinical condition to progress with the growth of the dog.

DCRV is a rare congenital cardiac disease in dogs, and details of its epidemiological and morphological characteristics have not previously been described. In this study, we obtained a number of findings, including the observation that DCRV occurs in small breeds of dog as well as in large breeds of dog, the possibility of a higher incidence in males, the existence of two types of DCRV in dogs, the presence of a higher incidence of other concurrent cardiac abnormalities, the possibility of concurrent TR being either congenital or acquired, and the fact that the DCRV is a congenital disorder although the clinical condition progresses with the growth of the dog. By conducting a long-term follow up on as many clinical cases as possible, we hope to be able to report on results of research with a focus on the treatment of DCRV in dogs.

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