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REVIEW

Surgical Interventions of Common Congenital Heart Defects in Dogs: A Comprehensive Review

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Summary

The most common congenital heart defects (CHDs) in the dog include aortic stenosis, patent ductus arteriosus, pulmonic stenosis, ventricular septal defects, mitral valve dysplasia, tricuspid valve dysplasia, tetralogy of Fallot, and endocardial fibroelastosis. They can occur as a result of genetic, environmental, infectious, or poisoning conditions, or malnutritional or maternal medical influences. Some authors believe that CHDs show gender and breed heritable predilections in dogs. Most dogs with severe CHDs may show congestive heart failure, and lead to an early death. The diagnoses of CHDs rely on electrocardiogram, chest radiograph, echocardiogram, and sometimes cardiac catheterization. Earlier accurate diagnosis with further proper treatments ensures better outcomes. Surgical interventions that are employed in humans can be applied in dogs as well. Open heart surgery may be necessary for the repair of the defects, but it costs much. Some CHDs may be curable to minimally invasive or hybrid procedures. Early diagnosis also allows owners to avoid continuing genetic defects in breeding lines. Attentions to the dog breeds with a known predisposition of an inheritable heart disease would merit a long-run veterinary significance.

Keywords: *Cardiac surgical procedure, Cardiopulmonary bypass, Septal occluder device, Veterinary clinics*

Köpeklerde Yaygın Konjenital Kalp Hatalarında Cerrahi Müdahaleler

Özet

Köpeklerde en yaygın konjenital kalp hataları (CHD) aort stenozu, açık duktus arteriozus, pulmoner darlık, ventriküler septal defekt, mitral kapak displazisi, trikuspital kapak displazisi, Fallot tetralojisi ve endokardiyal fibroelastozistir. Bu hatalar genetik, çevresel, enfeksiyöz veya zehirlenme durumları ile maternal malnutrisyon veya tıbbi etkiler sonucu oluşabilir. Bazı yazarlar CHD'lerin cinsiyet ve tür bazlı kalıtsal eğilime sahip olduğuna inanmakadır. CHD'li çoğu köpek konjestive kalp yetmezliği gösterir ve bunlar erken ölüme neden olur. CHD'nin teşhisi elektrokardiyogram, göğüs radyografisi, ekokardiyogram ve bazen kardiyak kateterizasyona dayanmaktadır. Erken doğru teşhis ve uygun tedavi daha iyi sonuçlar için gereklidir. İnsanlarda uygulanan cerrahi müdahaleler köpeklerde de uygulanabilir. Açık kalp ameliyatı hataların düzeltilmesi için gerekli olabilir ancak bu müdalae oldukça pahalıdır. Bazı CHD'ler invaziv ve hibrid yöntemler kullanılarak tedavi edilebilir. Erken teşhis hayvan sahiplerine sürülerinde genetik hatanın devam etmesini engellemede yardımcı olabilir. Bilinen kalıtsal bir kalp hastalığına yatkınlığı olduğu tespit edilen köpeklerin dikkatli takibi veterinerlik açısından uzun vadede önem kazanmaktadır.

Anahtar sözcükler: *Kardiyak cerrahi müdahale, Kardiyopulnomer bypass, Septal oklüzyon cihazı, Veteriner klinikleri*

INTRODUCTION

Congenital heart disease (CHD) represents a broad spectrum of heart defects at birth, single or combined, including those of the heart valves, cardiac chambers, great vessels, or abnormal connections between cardiac chambers [1], which may develop as genetics being the major causative etiology over environmental, infectious, or poisoning conditions, or maternal malnutritional or medical influences [2]. It is believed that CHDs show gender and breed heritable predilections in dogs [1]. In a study of a total of 1,132 heart defects in dogs, the incidence of CHD was 21.7%, with single defects accounting for 85%, 2 concurrent defects 14%, and 3 concurrent defects 1% [3]. Epidemiologic studies revealed that the most common CHDs were pulmonic stenosis, subaortic stenosis, and patent ductus arteriosus (PDA), followed by ventricular septal defect (VSD), valvular aortic stenosis, and tricuspid dysplasia [3,4]. Of them, subaortic stenosis, pulmonic stenosis, and VSD are frequently associated with other defects [3].

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Dogs with CHDs show varying symptoms with severity of the defects. Most dogs with severe CHDs show acute congestive heart failure, and lead to an early death [5], while dogs with mild-to-moderate CHDs may exhibit exercise intolerance, fainting episodes, and retarded growth, or even asymptomatic with only audible heart murmur on physical examination. They are easily managed and often have a good prognosis [6]. The diagnoses of CHDs rely on electrocardiogram, chest X-rays, echocardiogram and sometimes cardiac catheterization. Echocardiography has largely replaced cardiac catheterization as a routine diagnosis of CHDs in dogs due to accuracy and non-invasiveness [6]. With the speedy development of therapeutic strategies for CHDs, in particular, the development of minimally invasive surgical techniques in humans, CHDs in animals present more challenging management problems than before. The purpose of this article is to present a comprehensive literature review with regard to updated surgical interventions of the common CHDs in dogs of the current era.

PATENT DUCTUS ARTERIOSUS

PDA is the most common CHD in dogs, usually representing a left-to-right shunt [7]. It is more often seen in female than in male dogs, and in purebred than in mixed breeds [8]. The severity of the symptoms is closely related to the ductal size and aortopulmonary shunting [6]. Some animals remain asymptomatic for a long time and the cardiac murmurs are overlooked; others can manifest congestive heart failure as a result of pulmonary overperfusion. In right and left parasternal short-axis views of echocardiography at the level of the major vessels, the PDA presents as a hypoechoic space between the pulmonary arterial trunk and the aorta [9]. The angiographic studies on 43 dogs revealed minimal PDA diameter was 3.72±1.59 mm, and the diameter of the PDA ampulla was 8.46±3.01 mm. The frequency of PDA visualization was 78%, and the measurement deviation was 1-2 mm [10]. Good visualization of PDA by two-dimensional echocardiography may be achieved in 96% of the dogs [9]. Atrial fibrillation, mitral valvular incompetence, papillary muscle displacement and mitral valve prolapse are recognized complications of PDA [10]. If the PDA is left untreated, only 36% of dogs survive to one year of age. Thus, permanent occlusion at an early age is the current treatment strategy for PDA. Techniques for PDA repair include surgical ligation via thoracotomy and catheter-based occlusion. Surgical correction involves a left lateral thoracotomy and careful dissection around the PDA with circumferential ligatures. Thoracotomy ligation is an effective procedure with a success rate of up to 95% and a mortality rate of less than 2%. Complications such as fatal hemorrhage might occur due to increased ductus friability in particular in the older dog [11]. Catheter-based occlusion techniques are based on the use of coils, an Amplatzer plug, or an Amplatzer duct occluder. Preoperative angiography and transesophageal echocardiography are important means for the determination of the size and anatomy of the PDA [12]. The deployment of a detachable coil is usually under fluoroscopic guidance for locating correct placement for coils and the potential effectiveness of the occlusion [13]. The Amplatzer vascular plug is a self-expandable, cylindrical device attached to a delivery cable, and is usually delivered transvenously [14]. The choice of the Amplatz Canine Duct Occluder (ACDO) device size is 1.5-2 times of minimum ductal diameter based on echocardiographic or angiographic measurements. The procedures are comprised of advance of sheath and the device across the PDA into the main pulmonary artery, detachment of the device and subsequent retraction of the delivery cable from the descending aorta [15]. Tanaka et al.[16] informed that residual shunt was observed three months postoperatively and then a supplemental coil was inserted. Signh et al.[17] applied 4 kinds of devices ACDO (transarterial), Gianturco or MReye Flipper Detachable Embolization (Flipper) coil (transarterial), Amplatzer Vascular Plug (AVP) (transarterial), and Flipper coil (transvenous) for the management of PDA in dogs, and found ACDO had significantly fewer complications comparing with the other three. Saunders et al.[18] reported that the incidence of device migration was 3%. The ACDO mismatch can sometimes occur [19].

SUBAORTIC STENOSIS

Subaortic stenosis is a cardiac disorder with a narrowing of the descending aorta below the left ventricular outflow tract. The dog breeds such as Newfoundland are with more common subaortic stenosis which usually leads to an early death [20]. Subaortic stenosis has been genetically evidenced as an autosomal inheritance in Newfoundland dog [20]. A study on 195 untreated dogs with subaortic stenosis showed that sudden death was associated with severe subaortic stenosis 16 times more than moderate or mild, which often developed within the first three years of life [21]. Echocardiographic findings of 32 dogs with PDA were characterized by left ventricular hypertrophy in 37.5% (12/32), aortic insufficiency 62.5% (20/32) and subvalvular ridge 62.5% (20/32) [22]. The technique of cutting balloon valvuloplasty combined with high pressure valvuloplasty for dogs with severe subaortic stenosis has recently been reported to be a safe and feasible therapeutic option [23].

PULMONIC STENOSIS

Pulmonic stenosis is the third most common CHD in dogs. In pulmonic stenosis, the right ventricular outflow tract is narrowed at valve, supra-valve, or sub-valve levels, with the pulmonary valve stenosis being the most common

form. Minors et al.[24] studied infundibular pulmonic stenosis and further classified it into 3 subtypes: a fibrous diaphragm, fibromuscular, and muscular obstruction. Some animals with pulmonic stenosis manifest fatigue, fainting spells, ascites, exertional cyanosis, and even sudden death. Echocardiographic examinations reveal right ventricular concentric hypertrophy correlating to the severity of the pressure load imposed on the proximal right ventricular chamber by the stenosis. Performing balloon valvuloplasty reduces the risk of sudden death and improves quality of life as well. In the early years, balloon valvuloplasty was performed in 2 dogs, and immediate hemodynamic improvement was achieved with significant decrease of peak systolic pressure gradient across the pulmonic valve [25]. It was then evidenced that balloon valvuloplasty caused sustained right ventricular pressure reduction until 3 months after the intervention [26]. Minors et al.[24] performed surgical dilation of infundabular pulmonic stenosis without the need of cardiopulmonary bypass and systemic venous inflow occlusion. The dilated infundibular chamber was incised through a pursestring by a pediatric valve dilator across the stenosis for stricture relief. The peak pressure gradient was remarkably reduced 24 h after surgery than preoperation.

ATRIAL SEPTAL DEFECT

Atrial septal defect (ASD) represents 0.7% of the CHDs in dogs. The incidence of ASDs in dogs is rather high representing the second most commonly diagnosed CHD after mitral valve dysplasia [27]. Common signs associated with ASD include exercise intolerance, syncope, dyspnea, cough, heart murmur, cyanosis, and ascites if right heart failure develops. Medical treatment consists of systemic arterial vasodilation to reduce the shunt flow. Diuretics, angiotensin converting enzyme inhibitors and positive inotropic drugs are necessary for the animals with congestive heart failure, often resulting from severe aortic insufficiency [26]. Surgical repair of ASDs may be necessary in selected cases, but it costs much. A patch closure under cardiopulmonary bypass is a definitive treatment of ASD with large left-to-right shunting [28]. An alternative surgical operation is pulmonary artery banding for enhancing pulmonary artery resistance [6]. For some secundum-type ASDs, an Amplatzer device can be an alternative to close the defect. Amplatzer devices can be deployed by a right jugular or a transatrial approach through a right lateral thoracotomy [29]. Transcatheter ASD closure was successful in 10/13 dogs. After ASD closure, transthoracic color Doppler echocardiography indicated complete occlusion in 5 (50%) dogs, trivial to mild residual shunting in 4 (40%) dogs, and moderate residual shunting in 1 (10%) dog. Accidental right atrial release and embolization might occur in a few dogs. The mean event-free survival of the dogs with successful transcatheter ASD closure was 22.2±10.2 months.

VENTRICULAR SEPTAL DEFECT

Most VSDs in small animals are small and restrictive [30], moderate-sized VSDs are only partially restrictive with high right ventricle pressure, and large VSDs are nonrestrictive with a right ventricular pressure as high as systemic blood pressure [31]. Moderate and large defects impose an increased pressure load upon the right ventricle. Some animals can be asymptomatic [32]; others can be symptomatic with dyspnea, exercise intolerance, fainting, and cough. In severe cases, dogs may show congestive heart failure [33]. Diagnostic imaging can be helpful for the diagnosis of a VSD. Large shunting VSDs can be surgically repaired under cardiopulmonary bypass, and moderate or large shunting VSDs may also undergo pulmonary artery banding as a palliative procedure. Alternatively, the heart is approached by a right thoracotomy through the fifth intercostal space. By a ventricular incision, the VSD is closed primarily by an interrupted mattress suture with 6-0 pledgeted polypropylene. Simple closure of the VSD can effectively relieve of the associated aortic regurgitation, especially in case of the highly-located VSD. Early VSD closure may restrain the progression of the associated aortic valve regurgitation and minimize the risk of bacterial endocarditis [34]. In a study, a dog underwent a VSD repair with continuous sutures under cardiopulmonary bypass via a median sternotomy approach [35]. It was illustrated that right atrial incision was superior to right ventricular incision for the surgical repair of VSD under cardiopulmonary bypass in dogs, in particular, with better outcomes during postoperative recovery because of a shorter recovery period [36]. Besides, a secondary Gerbode defect due to infective endocarditis of the aortic valve was once reported in a 6-year-old intact male Great Pyrenees dog. Color Doppler revealed turbulent flow originating from the left ventricular outflow tract entering into the right atrium and right ventricle. Due to the severity of lesions and poor condition, the owner elected humane euthanasia and consented to necropsy without performing an operation [37]. In a dog with concurrent pulmonic stenosis, an Amplatzer occluder was used to successfully close the muscular type VSD [38].

ENDOCARDIAL CUSHION DEFECT (COMPLETE FORM)

The endocardial cushion defect (complete form) is composed of 6 leaflets: 3 left leaflets (left superior, left lateral, and left inferior) and 3 right leaflets (right superior, right lateral, and right inferior). Rastelli et al. classified complete endocardial cushion defects into types A, B, and C based on the morphology of the left superior leaflet and chordal attachment. Type A occurs most frequently, where the left superior leaflet is located above the left ventricle and attached to the crest of the VSD. Type B is

rare, where the left superior leaflet chordae are attached to anomalous papillary muscles from the inlet VSD in the right ventricle. Type C is characterized by marked bridging of the inlet VSD to the right ventricle by the left superior leaflet [39]. The complete form progresses more frequently into pulmonary hypertension compared with other CHDs. In children with complete endocardial cushion defect, irreversible pulmonary vascular damage occurs after 6 months of age, and surgical repair should be performed at 3-6 months of age. In dogs, timing of surgical intervention can be referred to that of the human. Two techniques are commonly used in people for repair of complete endocardial cushion defect: one-patch and two-patch techniques. In comparison to one-patch technique, two-patch technique in dogs is more effective in improvements of tricuspid regurgitation by avoiding postoperative residual shunting, and reducing operative mortality, incidence of arrhythmia, and re-intervention rate. A dog with endocardial cushion defect survived free of cardiac symptoms for 6 years and 5 months after two-patch technique repair under cardiopulmonary bypass [40]. The modified "Australian" technique [41] and no patch technique [42] require shorter aortic crossclamp and cardiopulmonary bypass times. Both are novel techniques for the surgical repair of complete endocardial cushion defect in human with good clinical results.

Two-patch technique [43]: A Gore-Tex patch is used to close the ventricular component of the defect and the atrioventricular valves are suspended to the top of the Gore-Tex patch. Another pericardial patch is used to close the atrial component and is sutured to the confluence of the atrioventricular valve and the Gore-Tex VSD patch. The mitral valve is sandwiched between the ventricular Gore-Tex patch and the atrial pericardial patch so that mitral valve laceration and possible dehiscence can be avoided.

Traditional one-patch technique [41]: After testing the competence of the common atrioventricular valve, a single approximating suture is placed over the septal crest between the superior and inferior leaflets. A Dacron patch is sutured to the middle of the septal crest by 5-0 pledgeted polypropylene sutures. The atrioventricular valve leaflets are anchored onto the Dacron patch. The mitral valve cleft is closed by interrupted polypropylene sutures. The atrial component of the defect is closed by a continuous suture placed in the remaining part of the patch, leaving the coronary sinus draining into the right atrium.

"Australian" one-patch technique [41]: This technique is different from the traditional one-patch technique in details of the surgical maneuver. After testing the competence of the common atrioventricular valve, the valve cleft is repaired using polypropylene sutures. The ventricular component of the defect is closed using pledgeted polypropylene sutures on the right ventricular aspect of the septal crest. Sutures are placed below the septal crest to avoid conduction tissue damage. The sutures are passed through the superior and inferior bridging leaflets making a partition boundary between the atrial outlets, and then passing through the Dacron patch. When tying the sutures, the ventricular component of the defect is obliterated. The Dacron patch was taken to close the atrial component of the defect by continuous sutures, leaving the coronary sinus draining into the right atrium.

No patch technique [42]: This technique is mainly pledgeted interrupted sutures anchoring on the right side of the VSD crest, passing through the midportion of the bridging atrioventricular leaflet. The left atrioventricular valve cleft is closed using interrupted sutures, and the ASD is closed by passing the VSD-closing interrupted sutures through the superior rim of the ostium primum defect, obliterating the VSD and ASD components by pulling the atrioventricular valve leaflets and atrial septum down to the VSD crest. The defects are closed without the use of a patch. A sliding plasty of the right atrioventricular valve septal leaflet is performed to improve the competence of the valve. In children, no-patch technique for repair of complete endocardial cushion defect resulted in no early deaths. This technique can surely be applied in animals with the advantages of shortened operation time.

TETRALOGY OF FALLOT

The incidence of tetralogy of Fallot is rarer ranging from 0.6% to 7% [44]. Diagnostic imaging may show a dextro-positioned and over-riding aorta, pulmonary valvular stenosis, ventricular and atrial septal defects, and right ventricular hypertrophy [45]. Conservative treatment includes diuretics, ß-blockers, and angiotensin-converting-enzyme inhibitor for relieving clinical symptoms. Oxygen therapy, puncture, and removal of fluids from the pleural and abdominal cavities may be necessary for deteriorated cases [44]. Surgical operations are an effective treatment. A complete correction of tetralogy of Fallot can be performed using a transatrial approach with limited ventriculotomy under cardiopulmonary bypass. The surgical procedures consist of hypertrophied infundibulum resection, primary closure of the VSD and right ventricular outflow tract reconstruction with a transannular pericardial patch. Transpulmonic pressure gradients were remarkably reduced at a 4-month follow-up [46]. Successful surgical repair can completely relieve the clinical signs [47]. A "side-by-side" type or modified Blalock-Taussig shunt between the subclavian artery to the pulmonary artery may increase pulmonary flow by systemic-pulmonary anastomoses and obliterating the animal's symptoms [44]. Surgical techniques of modified Blalock-Taussig shunt in human and animals have been described [48]. The balloon catheter dilation of the pulmonic stenotic lesion is a palliative procedure for animals with tetralogy of Fallot that are intolerable to anesthesia and open heart surgery. The balloon catheter is introduced into the

stenotic lesion under fluoroscopy for rapid and repeated inflations of the pulmonic infundibular stenosis. Post-dilation hemodynamic and angiographic evaluations revealed relief of pulmonic infundibular stenosis and increased pulmonary blood flow with no complications secondary to thoracotomy or long-term hospitalization [49].

COR TRIATRIATUM

Cor triatriatum dexter is a rare CHD in dogs, in which the right atrium is devided into two parts by a fibromuscular structure [50]. Affected dogs show signs of congestive heart failure including increasing exercise intolerance [51], severe cyanosis, dyspnea [52], tachypnea [53], progressive ascites [51], hepatomegaly [6], and growth retardation [54]. However, the diagnosis of cor triatriatum dexter is often delayed until the animal presents signs of right heart failure or a cardiac murmur. Nowadays, echocardiography is the diagnostic method of choice that has facilitated the definite diagnosis of cor triatriatum dexter. Cardiac catheterization can be helpful for the definitive diagnosis and hemodynamic evaluation. Medical management with enalaprild, furosemidee, and digoxin may transiently relieve congestive heart failure. Surgical correction under cardiopulmonary bypass is a reliable method for the treatment of cor triatriatum dexter [55]. Surgical repair of cor triatriatum dexter by a right lateral thoracotomy through the fifth intercostal space has been described [55]. The partitioning membrane was excised using Potts scissors and the inflow tract was expanded by a right atriotomy approach. Many animals experience an uneventful post-operative recovery [54,55], but surgical operation was declined in some animals due to associated complex abnormalities [50]. Balloon dilation has been applied in dogs with cor triatriatum dexter, and lead to improved clinical signs [51]. However, the partitioning membrane is often too fibrous to be easily ruptured by a balloon. Cutting balloon dilation was accordingly developed and successful relief of clinical signs including ascites in dogs has been reported [56]. Several balloon dilation procedures may be required to achieve permanent reduction of the obstructive pressure gradient across the membrane and resolution of clinical symptoms [57]. Therefore, despite non-invasiveness and effectiveness in some cases, balloon dilation is not always suitable for dogs and seems to be less reliable than surgical correction under cardiopulmonary bypass [55].

EBSTEIN'S ANOMALY

Ebstein's anomaly is a rare CHD of dog. This anomaly may be associated with ASD. Dogs may present exercise intolerance, dyspnea, cardiac murmur, and even ascites [58]. Echocardiogragraphy is helpful in reaching a definite diagnosis by illustration of right atrial enlargement and displacement and insufficiency of the tricuspid valve [59]. Oral administration of digoxin, vasodilators and diuretics partially improves the clinical symptoms, but sometimes cardiovascular deterioration and even sudden death may occur [58,60]. Recent veterinary literature has not shown successful surgical treatment for Ebstein's anomaly. However, it is believed that veterinarians may draw on the experience of relevant surgical techniques applied in human. Dogs with Ebstein's anomaly have poor prognosis. A dog suddenly died shortly after the diagnosis of Ebstein's anomaly was made, and another dog recovered better at the beginning, however, sudden death occurred later. Postmortem examinations showed right atrioventricular enlargement with thickened tricuspid leaflets [59].

MITRAL VALVE DYSPLASIA

Mitral valve dysplasia is a congenital malformation of the mitral valve complex representing 8% of the CHDs in dogs [4]. Canine breeds predisposing to develop mitral valve dysplasia are Bull Terriers, German Shepherds, and Great Danes [61]. Mitral valve dysplasia results in mitral valve regurgitation. Any component of the mitral valve complex (valve leaflets, chordae tendineae, and papillary muscles) may be malformed, and often more than one component is affected [62]. In addition, mitral valve dysplasia can be present as a member of a trilogy of defects (ASD, mitral valve dysplasia, and subaortic stenosis), which was once reported in a 4-year-old male castrated English bulldog who manifested exercise intolerance, multiple episodes of syncope, and a grade IV/VI heart murmur [27]. Dogs with mitral valve dysplasia often manifest congestive heart failure and a systolic cardiac murmur at the apex. Echocardiography discloses anterior mitral leaflet cleft resulting in left ventricular outflow tract obstruction [63]. The cleft divides the anterior leaflet into two parts, both of which extend across the subvalvular left ventricular outflow tract, and attachs to the subaortic interventricular septum [63]. White et al.[64] reported a dysplastic mitral valve dog case presenting with a history of collapse on exercise. Surgical intervention by open resection of the dysplastic mitral valve replaced with a bioprosthetic valve through a median sternotomy under cardiopulmonary bypass. The dog had a full recovery postoperatively. Behl et al.[65] reported successful mitral valve replacement in a beating heart, with resolved mitral regurgitation and improved cardiac performances.

TRICUSPID DYSPLASIA

Tricuspid dysplasia results in mostly tricuspid insufficiency and occasional tricuspid stenosis, which accounts for approximately 7% of all CHDs in dogs [4]. Tricuspid valve dysplasia has been reported in numerous dog breeds including old English sheepdogs, great Danes, German shepherds, and Irish setters [66]. Loud systolic heart murmurs can be a predomiant clinical manifestation [67]. Elongated and redundant tricuspid valve leaflets, thick,

shortened or even absent chordae tendineae, and severely dilated and malpositioned right atrium are common [68]. Associations with mitral valve dysplasia, septal defects, subaortic stenosis, pulmonic stenosis, situs inversus totalis [30] or Ebstein's anomaly [56] are rare, but can be present. Echocardiography demonstrates tricuspid valve malformations with severe dilations of the right atrium and ventricle, and Doppler echocardiography demonstrates severe tricuspid regurgitation [66]. Periodic thoracocentesis and/or abdominocentesis may be necessary [66]. Diuretics, vasodilators, digoxin, and an angiotensin converting enzyme inhibitor may also be indicated. Arai et al.[69] reported good intermediate-term outcomes in dogs with tricuspid vavle dysplagia undergoing tricuspid valve replacement with a bioprosthesis under cardiopulmonary bypass. Ten of the 12 (83.3%) dogs survived surgery and were discharged. Prosthesis-related complications include inflammatory pannus, thrombosis and endocarditis. Prognosis of tricuspid dysplasia can be poor if with associated cardiac abnormalities, and sometimes sudden death of the animals can occur [70].

COMMENTS

There are incidence and predilection discrepancies of CHDs between humans and animals. For example, tetralogy of Fallot occurs in 3/10.000 live births [71], which is much lower than the animal [44]. Animals with CHDs are likely to die prematurely. Valve dysplasias and large septal defects have a poor prognosis regardless of the method of treatment; whereas in humans, congenital valve dysplasia is not as predominant as in the animals, and the clinical outcome would be better. Therefore, affected dogs are often at a risk of congestive heart failure and sudden death. The diagnosis can usually be supported by echocardiography in addition to thoracic radiographic findings, etc. Early accurate diagnosis with further proper treatments may ensure the best outcomes. Interventional and/or surgical techniques that have been applied in humans are of considerable benefits to the animals with CHDs. Open heart surgery may be necessary to repair the defect, but it costs much. For CHDs, such as secundum-type ASDs, pulmonic stenosis, subaortic stenosis, and cor triatriatum dexter, etc., minimally invasive or hybrid procedures appear to be viable treatment options [70]. Early diagnosis also allows owners to avoid continuing genetic defects in breeding lines. Attentions to the dog breeds with a known predisposition of an inheritable heart disease would merit a long-run veterinary significance.

CONCLUSION

The occurrence of CHD in dogs shows breed and gender predilections. Definitive diagnosis of CHDs in dogs can be made by radiography, echocardiography and cardiac catheterization. CHDs in dogs may be curable by interventional or surgical treatment. Innovative surgical techniques especially minimally invasive procedures applied in humans may largely benefit the animals with CHDs.

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