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BEYOND DILATED CARDIOMYOPATHY: OTHER CANINE CARDIOMYOPATHIES

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ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is likely the second most common myocardial disease in dogs, with a high prevalence mainly in Boxer dogs (predominantly in the US) and English Bulldogs. This breed became predominant in some geographical areas. A small number of case reports on other breeds also detail an ARVC- like (or presumed ARVC) phenotype, but it is difficult to know for sure if this falls under ARVC or if it is just an ARVC like condition/ similar phenotype but different condition.

The definition of ARVC is taken from the human cardiomyopathy definitions: “Fibrous-fatty replacement of the normal myocytes, generally originating from the right ventricle (but not exclusive to the right ventricle) causing regional or global wall motion abnormalities, systolic dysfunction, arrhythmias and congestive heart failure”. In people, there is a shift in defining ARVC as simply Arrhythmogenic Cardiomyopathy, given that left ventricular involvement is more common than previously thought.

The disease is associated with genetic mutations affecting the intercalated discs, which are responsible for cell-to-cell adhesion. Similar changes have been noted in some Boxer dogs, so the disease is likely to be the same as the one described in people.

On gross pathology, the heart of dogs that died of ARVC can show minimal to obvious macroscopic changes, including dilated right ventricles with a pale myocardium, aneurismatic areas, wall thinning, and in some cases right chamber or generalised cardiomegaly. Right ventricular dilation has been reported to occur only in 30% of cases on a case series on ARVC in Boxer dogs, but it can be more frequent in English Bulldogs. On histopathology, the hallmark of the disease is the identification of fibro-fatty infiltration, which tends to be recognized more frequently on the right ventricle, starting from the epicardium towards the endocardium. The lesions are more commonly appreciated in the so-called “triangle of dysplasia” (right ventricular inflow, outflow tract and apex), but the lesions can also extend to the left ventricle and atria.

Diagnosis of ARVC

Because of the disease peculiar histopathology finding, the diagnosis of ARVC relies heavily on identifying these specific changes on histopathology. In veterinary medicine, given that endomyocardial biopsy is not routinely performed and similarly MRI is not available in most veterinary centres, the definitive diagnosis is currently based on post-mortem.

Most of the time we rely on a clinical diagnosis of ARVC, which takes into account clinical parameters (mainly ECG or Holter changes and echocardiographic characterization). This technically should be considered a “presumed ARVC” as the definitive diagnosis would require histopathology. We also lack histopathology characterization of most presumed ARVC cases in

Bulldogs, although it is reasonable to consider that it is likely the same presentation as humans and Boxer dogs.

Because the condition mainly affects two breeds, and the American Boxer dog population seems to be highly affected (mainly in the past years), it is reasonable to say that a Boxer dog with determinate ECG changes is likely affected by ARVC. However, there are a couple of issues worth considering. First of all, some dogs may develop or present with CHF and a DCM phenotype, and at present we do not currently know whether the DCM phenotype is the result of long-standing damage of the myocardium due to ARVC or whether some of these cases should be regarded as DCM/ DCM phenotype.
Secondly, the presence of ventricular premature complexes may be secondary to myocardial damage that has occurred due to other disease conditions (including myocarditis or systemic disease); although it would be unusual to see only left-bundle-branch-block-morphology VPCs in a dog with myocarditis, this could still be possible.

Clinical presentation

The clinical presentation of dogs with ARVC is variable, but similarly to dogs with DCM, it can be divided into a preclinical phase, whereby no clinical signs are noted but structural or arrhythmic alterations are present, and an overt clinical phase, whereby arrhythmia-related clinical signs or congestive heart failure (or both) are present. Similar to dogs with DCM, dogs with ARVC may be brought to visit for non-cardiac reasons, for non-specific deterioration (including weight loss, lethargy) or cardiac-related reasons (exercise intolerance, syncope, abdominal distension, tachypnoea or coughing). Some dogs may also experience sudden cardiac death as the first presenting sign.

Most dogs are middle age (5-7 yo), but older dogs may be affected as well.

As always, the disease is likely a continuum of clinical signs, so some dogs may present equally with both arrhythmia and congestive heart failure signs.

Here is a list of clinical presentation/ complaints from the three more numerous case series available in the literature:

Clinical presentation	Boxer	English Bulldog
Sudden cardiac death (incidence)	39% (mostly during exercise, but also asleep)	22%
Collapse/ Syncope	52-80% (mostly with echo changes)	38-90% (mostly asymptomatic)
Dyspnea	30-56 %	0-69% (mostly with CHF)
Weight loss	5-31%	Not reported
Cough	15-64%	44%

Treatment options for dogs with ARVC

The treatment of dogs with ARVC is aimed at arrhythmia management and CHF management.

Whilst in situations where CHF or systolic dysfunction is present, drugs' choice is probably more intuitive and in line with other disease conditions, there are no strict guidelines about arrhythmia management. This entails the decision about when to treat and also which

antiarrhythmic drug to choose. A study evaluating four different antiarrhythmic protocols in Boxer dogs with at least 500 VPC/24hr found that two protocols were effective in reducing VPC count, but not syncopal events.

In people, treatment options include the implant of a cardiac defibrillator, which was tested in a dog but lead to inappropriate shocks due to programming/ sensing, so this option is not currently advised in dogs with ARVC.

Dogs with ARVC and arrhythmias

The main caveat when deciding to treat dogs with ARVC and arrhythmia is related to the fact that sudden cardiac death does not seem to be strongly correlated to the severity of Holter findings, mainly for those dogs that do not show increased malignancy/ complexity on Holter monitoring.
It is reasonable and ethical to offer cardiac treatment in those dogs that present a high number of ventricular ectopic beats and that present ventricular tachycardia or other complex ventricular rhythms (including couplets, triplets, bigeminal, trigeminal or quadrigeminal rhythms as well as R-on-T phenomenon), but for dogs that present a low number of VPCs, the decision to treat is not so clear-cut: this has to balance the need to decrease the risk with overtreating a patient with no clinical benefit.

HYPERTROPHIC CARDIOMYOPATHY (HCM) PHENOTYPE

An HCM phenotype can seldomly be observed in dogs, but the diagnosis of primary HCM is a diagnosis of exclusion and is even rarer. Most commonly, an HCM phenotype is secondary to cardiac or systemic conditions causing concentric LV hypertrophy.

Cardiac conditions:

- Subaortic stenosis
- Dynamic left ventricular outflow tract obstruction (+/- mitral valve dysplasia)
- Infiltrative disease (neoplastic infiltration)
- Myocarditis

Systemic conditions:

- Hypovolemia (“pseudohypertrophy”)
- Systemic hypertension
- Hyperadrenocorticism
- Obesity

<u>Dynamic left ventricular outflow tract obstruction in dogs</u>

This condition is uncommon, but it can cause secondary LV wall hypertrophy due to obstruction of the LV outflow tract, similar to subaortic stenosis but with a dynamic and not fixed component. LVOT obstruction can occur due to mitral valve abnormalities (mainly mitral valve dysplasia), can be present despite no obvious mitral or LVOT abnormalities, or can be induced by hypovolemia (altering mitral valve anatomy/ relationship with LV cavity).

Dogs may present for murmur investigation, syncopal events and some may also present congestive heart failure signs at presentation.

Typical echocardiographic findings include concentric LV hypertrophy (sometimes biventricular), systolic anterior motion of the mitral valve, increased aortic flow with a concave dagger-shaped acceleration profile typical of dynamic obstruction. Due to the presence of substantial LV wall thickening, diastolic function is impaired, and some degree of diastolic dysfunction is observed. The mitral valve may show elongated, thickened leaflets, increasing mitral to interventricular septal contact and favoring systolic anterior motion (or possibly also chordal anterior motion) or causing LV morphological changes that favor dynamic left ventricular outflow tract obstruction.

Some of these changes were reversible by administration of β-blockers and by no treatment in two additional cases. It would probably be reasonable to offer treatment, alongside with ruling out any systemic condition that could favor onset of LV wall hypertrophy (including systemic hypertension)

Mitral valve dysplasia and an associated HCM phenotype was frequently reported in Dalmatians and Rottweilers. Pointers dogs and Terrier breeds seemed also to be affected by LV wall hypertrophy with dynamic left ventricular outflow tract obstruction, but no evidence of obvious abnormalities of the mitral valve or papillary muscle suggesting mitral valve dysplasia were identified, so that the mechanism for dynamic left ventricular outflow tract obstruction may be different in these breeds. Another uncommon situation of dynamic left ventricular outflow tract obstruction is due to a mass in the subaortic area.

Hypovolemia or right-sided pressure overload resulting in an underfilled left ventricle can both favour geometrical LV remodeling, with the end-result being systolic anterior motion of the mitral. This is likely a temporary finding if the primary cause for hypovolemia/ right-ventricular pressure overload is successfully treated.

MYOCARDIAL ISCHEMIA/ INFARCTS

Myocardial ischemia is characterised by decreased blood supply, while infarction is the cellular response to lack of perfusion, characterised by cellular death.

Acute or chronic myocardial infarctions are uncommon in dogs and appear to be associated with concurrent systemic or cardiac disease that led to a thromboembolic state. Most of the reported cases are, as a matter of fact, post-mortem. Conditions associated with myocardial ischemia include endocarditis, neoplasia, renal disease, immune-mediated hemolytic anemia, hypothyroidism and pancreatic disease. Rarely myocardial infarctions are the consequence of coronary artery disease/ coronary artery spasms due to atherosclerosis or congenital abnormalities of the coronary arteries.

Myocardial infarcts and ischemic cardiomyopathy in dogs

Reversible myocardial ischemia with a stunned myocardium can occasionally be observed in dogs undergoing emergency abdominal surgery (extra-cardiac causes) that is accompanied by severe hypovolemia/ hypotension. Arrhythmias may also accompany hypokinetic, dyssynchronous hearts, so wall motion abnormalities may be present, but the underlying cause

can be more difficult to be ascertained. Some of these dogs tend to show runs of accelerated idioventricular rhythms postoperatively, which are self-limiting and do not require treatment.

There is no extensive data about systemic conditions associated with myocardial stunning and wall motion abnormalities in dogs, but in the author's experience it is likely the most common clinical scenario of myocardial ischemia in dogs.

The available data of myocardial infarction in dogs is scant. A retrospective, post-mortem study identified 32 dogs undergoing post-mortem evaluation over 9 years. Electrocardiographic abnormalities included ventricular tachycardia (16%), atrial fibrillation (9%), and premature ventricular contractions (6.5%). On echocardiography, dilated, poorly contractile hearts (n = 3) and thickened mitral valves (2) were identified. The majority of dogs (97%) had multi-vessel infarction affecting the left ventricle as well as other other portions of the heart.

Other findings potentially linked to myocardial ischemia and infarction on post-mortem include arteriosclerotic lesions. Arteriosclerosis is characterised by chronic luminal narrowing from proliferative and degenerative non-inflammatory changes in the vessel wall, in contrast with atherosclerosis, which is characterised by fatty degeneration of extramural coronary arteries.

A retrospective, study identified arteriosclerotic lesions in 65 dogs undergoing necropsy, mainly old, large breed dogs that presented for cardiac complaints (including sudden cardiac death). The majority of the cases died due to congestive heart failure (46%), followed by sudden cardiac death (20%) and intra- or postoperative death or euthanasia (15%). Echocardiographic studies prior to death identified hypokinesia (either subjective or by objective quantification) in most dogs (79%). Well demarcated foci of necrosis and fibrosis were observed in the ventricular myocardium of all dogs and were often localised to the papillary muscles. Valvular endocardiosis was observed in a third of the cases and none of the dog included in the study was classified as affected by DCM. The clinical impact of these findings is unknown in dogs, as the study was aimed at presenting a collection of cases with specific post-mortem findings, but the actual prevalence of arteriosclerosis in the overall population of dogs undergoing necropsy was not provided. Similarly, no readily available clinical test or echocardiographic parameters are available to correlate the histopathological findings to the clinical cases.

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