Arrhythmogenic right ventricular cardiomyopathy in a dog

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ABSTRACT

An 8-month-old Labrador retriever bitch was evaluated for sudden-onset, progressive abdominal distension. Physical examination revealed an exaggerated inspiratory effort, severe ascites, bilateral jugular vein distension, and hypokinetic femoral arterial pulses. Thoracic auscultation detected tachycardia with muffled heart sounds, without audible cardiac murmurs. Thoracic radiographs identified severe right ventricular enlargement and pleural effusion. The electrocardiogram was consistent with incomplete right bundle branch block or right ventricular enlargement. Echocardiography demonstrated severe right ventricular and atrial dilation, secondary tricuspid regurgitation, and thinning and hypocontractility of the right ventricular myocardium. Left heart chamber sizes were slightly decreased, with normal left ventricular contractility. A diagnosis of arrhythmogenic right ventricular cardiomyopathy was reached, based on the characteristic clinical, electrocardiographic, radiographic and echocardiographic findings, and the exclusion of other causes of isolated right ventricular failure. Treatment effected good control of clinical signs, until acutely decompensated congestive right heart failure led to euthanasia after 4 months. Arrhythmogenic right ventricular cardiomyopathy is a well-described clinical entity in humans, and has previously been documented in 3 male dogs. The condition is characterised by progressive fibro-adipose replacement of right ventricular myocardium, while the left ventricle usually remains unaffected. It should be considered a differential diagnosis in any young dog presented with isolated right heart failure, syncope, or unexplained ventricular tachyarrhythmias. This article reports the 1st case of arrhythmogenic right ventricular cardiomyopathy in a female dog, and highlights its echocardiographic features.

Key words: arrhythmogenic right ventricular cardiomyopathy, canine, echocardiography, right bundle branch block.

Möhr A J, Kirberger R M **Arrhythmogenic right ventricular cardiomyopathy in a dog**. *Journal of the South African Veterinary Association* (2000) 71(2): 125–130 (En.). Department of Companion Animal Medicine, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a well-described clinical entity in humans[3,6,9,10,22,26]. The disorder is characterised by progressive fibro-adipose replacement of right ventricular myocardium, initially with regional and later global right ventricular involvement[6,8–10]. The left ventricle and atrium usually remain unaffected[6,9,22], distinguishing this condition from idiopathic dilated cardiomyopathy (DCM). The condition in humans is most prevalent in adolescent males, with syncope, congestive right-sided heart failure, or sudden death being the most common clinical manifestations[6,10]. The aetiology and pathogenesis (degenerative, infectious, inflammatory or proliferative)

remain to be established[9], although the role of apoptosis (programmed cell death) in right ventricular pathology has recently been proposed[21], and a familial incidence has been demonstrated in some instances[26]. Although it has been termed right ventricular dysplasia in some of the literature referring to humans[3,10,20,21], abnormal embryogenesis is not believed by all investigators to be involved[6,9], and the term cardiomyopathy may be more appropriate. Ventricular tachycardia (often of left or right bundle branch block configuration) or supraventricular arrhythmias occur in most human patients who have the condition[6,9,10,20,22]. Refractory right-sided cardiac failure or lethal arrhythmias are the usual cause of death, and prognosis is generally poor[6,10].

In animals, arrhythmogenic right ventricular cardiomyopathy has previously been described in 3 dogs[5,23,25], cats[8], and reportedly also the ferret[8]. The 3 reported canine cases were young to middle-aged males (Bull mastiff, Siberian husky and

dachshund), with acute-onset congestive right-sided heart failure[23,25] or syncope[5] as the presenting complaints. Cardiac arrhythmias (ventricular tachycardia of left bundle branch block configuration), in the absence of cardiac murmurs, was a common feature at the time of diagnosis[5,23,25]. Thoracic radiography in these dogs revealed right ventricular cardiomegaly[5], while echocardiography demonstrated severe right ventricular and atrial dilation[23,25], tricuspid regurgitation[5,23,25], normal left ventricular contractility, and normal to decreased left ventricular chamber sizes[5,23,25]. These radiographic[22] and echocardiographic[6,10,22] findings are also a feature of human ARVC. Survival times in the reported dogs ranged from a few days (euthanased)[25], to 3 weeks[5] and 3 months[23] (sudden death).

This article reports the 1st recognised case of ARVC in a female dog, and highlights the echocardiographic features of the disorder.

CASE HISTORY

An 8-month-old, 27 kg intact female Labrador retriever was referred to the Onderstepoort Veterinary Academic Hospital with a complaint of sudden-onset progressive abdominal distension of 1 week's duration. Physical examination revealed an exaggerated inspiratory effort, generalised muscle atrophy, and pale-pink mucous membranes, with a normal capillary refill time. The abdomen was severely distended, non-painful, and on percussion elicited a distinct fluid wave. Bilateral jugular vein distension and hypokinetic femoral arterial pulses with pulse deficits were also detected. On thoracic auscultation, tachycardia (220 beats per minute), slightly muffled heart sounds, and no audible cardiac murmurs were present. Right-sided congestive heart failure was suspected clinically.

Urine analysis revealed mild proteinuria (30 mg/dℓ) and a specific gravity of 1.023. Faecal analysis was normal. Abnormalities in the serum biochemical profile included a moderate decrease in total serum protein concentration (37.7 g/ℓ; normal 53–75 g/ℓ), due to both mild hypoalbuminaemia (25.6 g/ℓ; normal 27–35 g/ℓ) and moderate hypoglobuli-

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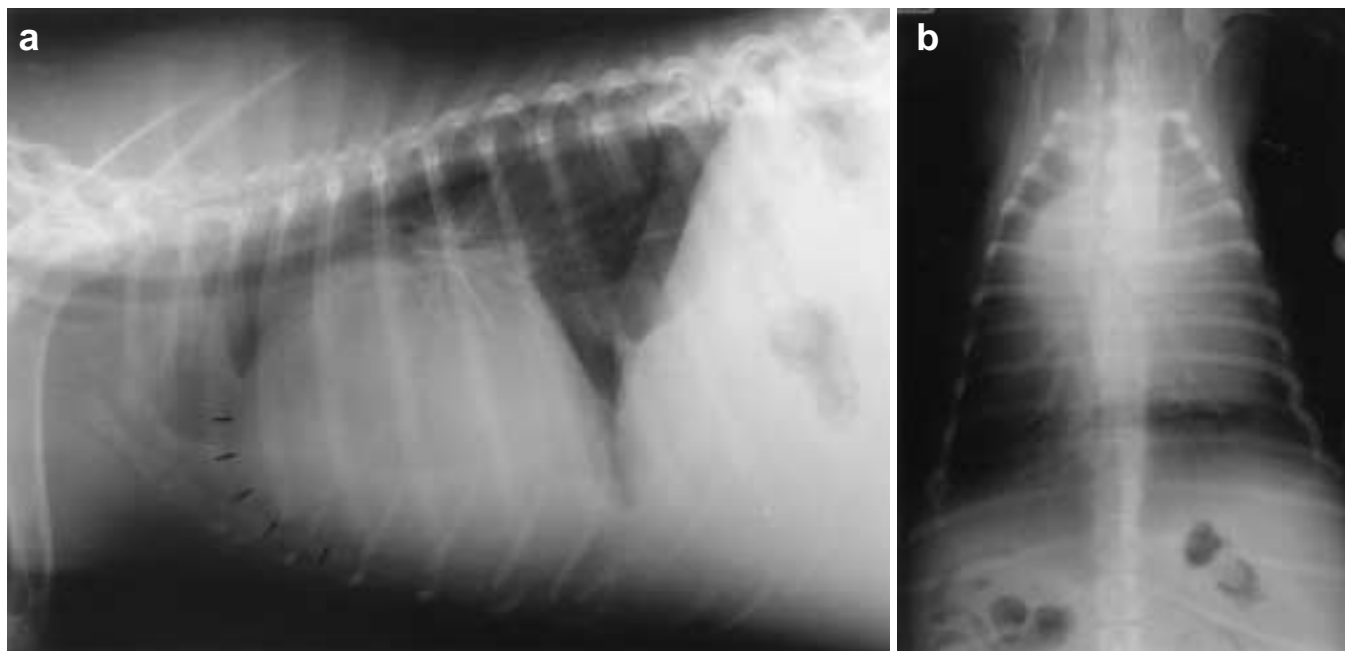
Fig. 1: a, **Right lateral recumbent thoracic radiograph, illustrating severe right ventricular enlargement** (arrows), **increased cardiac sternal contact, and elevation of the intrathoracic trachea. Signs of congestive right heart failure include distension of the caudal vena cava, pleural effusion, and ascites;** b, **dorsoventral thoracic radiograph, illustrating displacement of the cardiac apex to the left, while right ventricular enlargement is effaced by pleural effusion.**

naemia (12.1 g/ℓ; normal 20–37 g/ℓ); mild hyperkalaemia (5.3 mmol/ℓ; normal 3.6–5.1 mmol/ℓ) and hyperphosphataemia (1.8 mmol/ℓ; normal 0.9–1.6 mmol/ℓ). The complete blood count showed no significant abnormalities. Abdominal fluid analysis revealed a modified transudate (total protein 38 g/ℓ, specific gravity 1.023, 1100 nucleated cells/mℓ).

Survey thoracic radiographs (Fig. 1a,b) demonstrated a massively enlarged cardiac silhouette (vertebral heart size 14; normal <10.5)[2], leading to marked elevation of the intra-thoracic trachea cranial to its bifurcation. Increased cardiac sternal contact (Fig. 1a) with a cranial cardiac wall bulge, in combination with a greater increase in craniocaudal cardiac diameter as compared to apicobasilar diameter, suggested severe right ventricular enlargement. On the dorsoventral view (Fig. 1b) the cardiac apex was displaced to the left, while the expected right ventricular enlargement was effaced by pleural effusion. Visible signs of right-sided congestive heart failure included ascites, moderate pleural effusion, and caudal vena caval distension. The pulmonary vasculature appeared normal.

Electrocardiographic (ECG) findings included sinus rhythm with a small R wave amplitude, prominent S waves in leads I, II, III, and aVF, and a normal QRS complex duration (Fig. 2). Additionally, P-wave duration was increased, and high-amplitude T waves could be seen fusing with the QRS complexes.

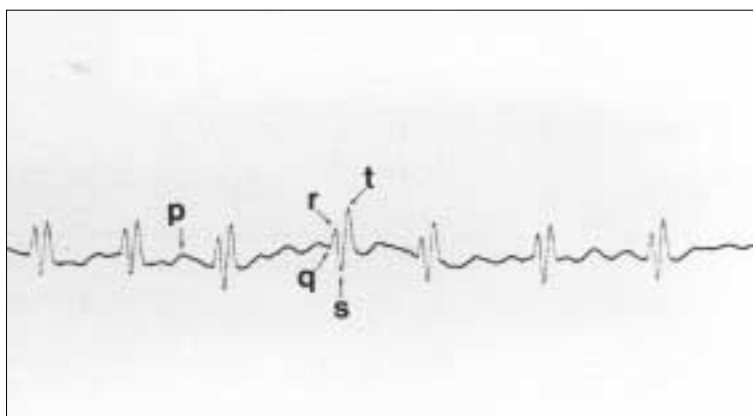Transthoracic echocardiography (Aloka

Fig. 2: **Lead II electrocardiographic recording, consistent with incomplete right bundle branch block or right ventricular enlargement. Note the abnormally low R wave amplitudes, prominent S waves, and normal QRS complex duration. Other abnormalities include increased P wave duration and high-amplitude T waves fusing with the QRS complexes.** Paper speed = 50 mm/s; 1 cm = 1 mV.

SSD-630 Echo Camera and Aloka Doppler unit UGR-38) was performed using standardised imaging planes and display conventions[27]. The expected appearance of the 2-dimensional right parasternal long-axis 4-chamber view was distorted as a result of severe right ventricular and atrial dilation (Fig. 3), with subjectively assessed thinning and hypocontractility of right ventricular free wall myocardium; the left heart appeared unaffected. The tricuspid valve's annulus diameter was widened, while valvular location and the leaflets themselves appeared normal. Spectral Doppler tracings on the atrial side of the tricuspid valve demonstrated high-velocity (175 cm/sec) turbulent systolic regurgitant flow into the right atrium, while all other heart

valves showed normal unidirectional laminar flow.

M-mode measurements (Table 1) obtained from the right parasternal window illustrated marked increases in right ventricular end systolic and end diastolic diameters, with moderately decreased left ventricular end systolic and diastolic diameters. Right ventricular fractional shortening (FS) was markedly decreased, while left ventricular FS was within the normal range.

Spectral Doppler demonstrated decreased peak flow velocities over all heart valves (Table 2). In addition to a decreased peak velocity, flow over the aortic valve showed a repetitive pattern of higher-and-lower velocities during consecutive cardiac cycles (Fig. 4), with

Fig. 3: **Right parasternal long-axis 4-chamber echocardiogram. The right ventricle** (RV) **and right atrium** (RA) **are markedly dilated, while left ventricular** (LV) **and atrial** (A) **chamber sizes are slightly decreased. The image is inverted compared to standard display conventions[27].**

the lower velocity peaks occurring in synchrony with femoral arterial pulse deficits.

In order to rule out an atrial septal defect as a cause of the right heart dilation, non-selective intravenous contrast echocardiography was performed by injecting 10 mℓ agitated saline into the left cephalic vein. The air bubbles could be followed as refractile specks entering the heart *via* the right atrium and filling the right ventricle. The presence of an atrial septal defect was eliminated, since no bubbles appeared in the left atrium, and no filling defect was present in the right atrium. A small (3 mm at septal base) anechoic filling defect could be visualised intermittently on the right ventricular

side of the membranous interventricular septum, representing a small interventricular septal defect.

Cardiac catheterisation was recommended to measure pulmonary arterial and right ventricular pressures, in order to definitively exclude cor pulmonale, and to possibly obtain an endomyocardial biopsy. The owners, however, declined any further diagnostic procedures.

A final diagnosis of ARVC was reached, based on the characteristic clinical, radiographic, ECG and echocardiographic appearance of the condition, and the exclusion of other causes of isolated right-sided cardiac dilation (*e.g.* tricuspid dysplasia, Ebstein's anomaly, tricuspid chordal rupture, pulmonary artery regurgitation and significant left-to-right intracardiac shunts).

Treatment for ARVC with congestive right-sided heart failure consisted of enalapril (Renitec, Merck Sharpe & Dohme) 0.5 mg/kg, per os (PO), once per day; furosemide (Puresis, Lennon) 2 mg/kg, PO, 3 times daily for the initial 5 days, followed by twice daily dosing; and digoxin elixir (Lanoxin, Glaxo Wellcome) 0.006 mg/kg, PO, twice per day. The digoxin dose was initially adjusted for the degree of ascites[14] and later according to serum concentrations of the drug. Although plasma amino-acid concentrations were not assayed, L-carnitine (Adam Todd Investments) 220 mg/kg, PO, once a day; and taurine (Kyron Laboratories) 1.25 g, PO, twice a day, were supplemented owing to their potential therapeutic benefit in other canine dilated cardiomyopathic states[12,14,18]. A diet moderately restricted in sodium was fed.

Four days later the heart rate had normalised (100 beats per minute), serum albumin concentration returned to within the normal range, abdominal effusion had markedly decreased (cranial abdominal girth decreased from 85 cm to 75 cm), and the dog had lost 5 kg in weight. She was discharged 7 days after admission.

Four months after the initial diagnosis of ARVC, during which time clinical signs had been well controlled by the prescribed treatment, the dog was again presented to the referring veterinarian for acute onset ascites and dyspnoea. The sudden deterioration was considered to be the result of acutely decompensated congestive right-sided heart failure, and the dog was euthanased at the owners' request. The owners elected not to have a necropsy examination performed.

DISCUSSION

Cardiomyopathy is a term used to describe cardiac disease conditions affect-

Table 1: **M-mode echocardiographic measurements obtained from the right parasternal window.**

	Patient	**Normal[1]**
LV ESD	22 mm	29–35.5 mm
LV EDD	31 mm	42.5–49.5 mm
LV FS	29 %	28–45 %
RV ESD	51 mm	14.5–17.7 mm
RV EDD	64 mm	21.2–24.7 mm
RV FS	17 %	28–45 %*
AO diameter	20 mm	23–27.5 mm
LA diameter	20 mm	19.5–25.5 mm
LA:AO	1	0.95–0.97

LV = left ventricle; ESD = end systolic diameter; EDD = end diastolic diameter; FS = fractional shortening; RV = right ventricle; AO = aorta; LA = left atrium.
*No published values for normal right ventricular fractional shortening could be found in the literature; however, values similar to normal left ventricular fractional shortening would be expected.

Table 2: **Spectral Doppler echocardiographic peak flow velocities.**

	Patient (cm/sec)	**Normal (cm/sec)[16]**
PV peak velocity	80	100–140
MV 1st peak velocity	40	76–106
MV 2nd peak velocity	30	50–76
TV 1st peak velocity	50	66–106
TV 2nd peak velocity	50	42–74
AV peak velocity	80/40*	124–190

PV = pulmonary valve; MV = mitral valve; TV = tricuspid valve; AV = aortic valve.
*Flow over the aortic valve showed a repetitive pattern of higher and lower velocities during consecutive cardiac cycles – see text for details.
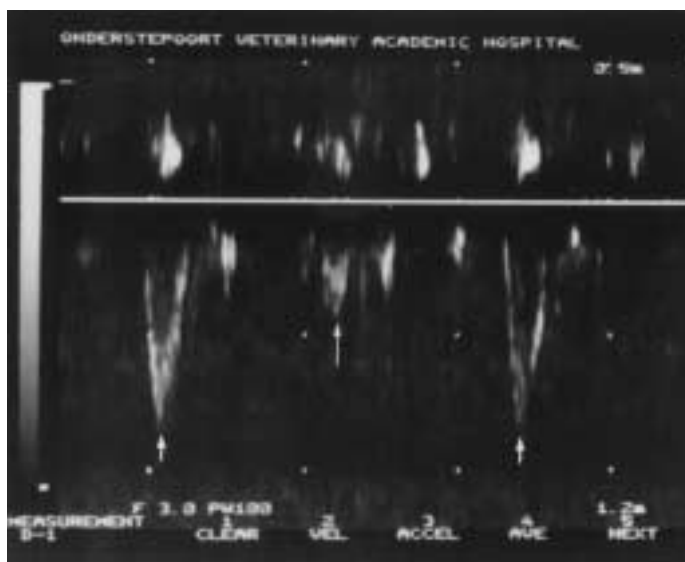
Fig. 4: **Spectral Doppler recording over the aortic valve, showing decreased peak systolic velocity** (flow below the baseline). **An alternating pattern of higher** (short arrows; 80 cm/sec) **and lower** (long arrow; 40 cm/sec) **velocities during consecutive systolic cardiac cycles is seen, with the lower velocity peaks occurring in synchrony with femoral arterial pulse deficits.**

ing the myocardium, as distinguished from pathology primarily involving the pericardium, cardiac valves, or congenital anatomic cardiac anomalies, which may all eventually lead to myocardial failure[11]. Secondary cardiomyopathies are the consequence of systemic or metabolic disease, whereas primary cardiomyopathies arise in the myocardium itself[11]. Idiopathic dilated cardiomyopathy is the most common form of primary cardiomyopathy in dogs, and virtually always involves the left ventricle, or both the left and right ventricles in combination[13,23]. Echocardiographic features of DCM include decreased left ventricular FS (systolic dysfunction) with resultant left ventricular and atrial dilation, and possibly secondary right heart dilation[13]. The case described in this report differs significantly from the above description of DCM in that the primary myocardial dysfunction involved the right heart, while left-sided cardiac contractile function remained spared. This resembles ARVC as described in humans[3,6,9,10,22,26] and animals[5,8,23,25].

Survey thoracic radiographs revealed massive right-sided cardiomegaly without pulmonary congestion. Congestive right-sided heart failure was reflected by a modified transudate ascites[4], pleural effusion, and distended jugular veins and caudal vena cava. Elevated hydrostatic pressure secondary to right heart failure, with the loss of both albumin and globulin into body cavity effusions, would account for the panhypoproteinaemia observed. The mild hyperkalaemia and hyperphosphataemia likely reflect decreased renal perfusion with a reduction in

glomerular filtration rate, while the hyperphosphataemia may also have been age-related[24].

Echocardiography clearly demonstrated severe dilation of the right ventricle and atrium, right ventricular hypocontractility, and a decreased left ventricular chamber size with normal contractile function. Right ventricular dilation classically develops as a sequel to either myocardial failure or ventricular volume overload conditions (increased preload)[15]. Reduced right ventricular (RV) contractility, such as occurs in ARVC, leads to right heart systolic ejection failure, with a resultant increase in RV end systolic volumes. Chronic RV volume overload, in combination with this primary myocardial failure, will lead to dilation of the chamber, subsequent widening of the tricuspid valvular annulus resulting in tricuspid regurgitation, and secondary right atrial dilation. Right ventricular contractile failure also leads to decreased blood delivery to the left heart, accounting for the reduction in left ventricular chamber sizes, and decreased flow velocities over the aortic, mitral and pulmonary valves seen in this case. Tricuspid peak velocity flow was greater than that over the mitral valve, due to tricuspid regurgitation and right heart volume overload. Similar to the previously reported canine cases of ARVC[5,23,25], no cardiac murmur was auscultated at the time of diagnosis. The absence of a murmur in the presence of tricuspid regurgitation indicates a large, patent regurgitant orifice. The normal echocardiographic appearance of the tricuspid valvular leaflets and normal tricuspid valve location ruled out

tricuspid dysplasia and Ebstein's anomaly (abnormal tricuspid valvular insertion)[22], respectively, as potential causes of the RV volume overload. Tricuspid chordal rupture or pulmonary artery regurgitation as causes of the RV volume overload were similarly excluded by echocardiography, while significant intracardiac shunts from the left heart chambers to the right atrium were excluded by contrast echocardiography. The presence of a small (3 mm) interventricular septal defect in this dog was considered an incidental finding unrelated to the severe cardiac pathology. Ventricular septal defects are common congenital cardiac abnormalities in dogs; small defects with mild left-to right shunts produce little haemodynamic change, with the dogs remaining asymptomatic[7]. Large defects with left-to-right shunting cause pulmonary circulation and left heart volume overload, with resultant pulmonary oedema and left heart dilation[7], which were absent in this case.

In contrast to the right ventricular dilation seen in myocardial failure or ventricular volume overload, conditions leading to pressure overload (increased afterload) of the right ventricle cause increased wall thickness (concentric hypertrophy) with normal or diminished ventricular dimensions and volume[15]. An exception to this rule occurs in acquired pulmonary hypertension with cor pulmonale, where right ventricular dilation may develop in combination with concentric hypertrophy[15]. Causes of pulmonary hypertension include chronic heartworm disease, severe parenchymal pulmonary disease, chronic alveolar hypoxia, and pulmonary thromboembolism. Cardiac catheterisation to measure pulmonary arterial and right ventricular pressures to definitively exclude cor pulmonale was not performed in this case. However, radiological signs of pulmonary hypertension (widening of proximal pulmonary arteries and tortuous middle arterial portions)[7] and echocardiographic signs (pulmonary artery dilation and right ventricular concentric hypertrophy), were absent. The dog also had no history or radiographic signs of chronic respiratory disease, and heartworm is not endemic to South Africa. Congenital (primary) pulmonary hypertension leads to pure right ventricular concentric hypertrophy[15] rather than the dilation seen in this case.

Apposition of the right ventricular endocardium to the epicardium, owing to loss of the myocardium (Uhl's anomaly), may also lead to isolated right-sided cardiac failure[21]. It has recently been speculated that human Uhl's anomaly and ARVC may be a spectrum of the same

disease process[21], and cases of both canine[5] and feline[19] Uhl's anomaly have been classified as ARVC. It has also been suggested that ARVC in humans may in fact represent a more generalised cardiomyopathy, with disproportionate right ventricular manifestations[6,10]. This possibility has also been suggested in cats[8] and dogs[28], and the left ventricular FS in the low-normal range (without left ventricular dilation) in the present case makes this an interesting possibility.

Doppler blood flow over the aortic valve showed a repetitive pattern of higher-and-lower flow velocities during consecutive cardiac cycles, with the lower velocity peaks occurring concurrently with femoral arterial pulse deficits. A suggested cause is that right ventricular contractile dysfunction (with decreased right ventricular systolic ejection) resulted in left ventricular underfilling, and consequently decreased left ventricular end diastolic volumes. This in turn would lead to a decreased stroke volume (Frank-Starling law) and aortic flow velocity, with a higher than expected residual end systolic left ventricular volume at the end of that cardiac cycle. During the next cardiac cycle, the blood volume delivered to the left ventricle will be added to the residual volume present from the preceding cardiac cycle, with resultant stronger inotropic contraction, stroke volume, and a higher velocity aortic flow. The forward failure thus created explains the pulse deficits and pale mucous membranes in this dog.

Ventricular tachycardia (usually of left or right bundle branch block configuration) is commonly diagnosed in humans[6,9,10,20,22] suffering from ARVC. The present case showed ECG abnormalities consistent with incomplete right bundle branch block or right ventricular enlargement. Incomplete right bundle branch block may potentially be the result of an increased right ventricular chamber size, leading to stretching or disruption of the moderator band[17]. Since right bundle branch blocks alone do not cause clinical sequelae[17], no anti-arrhythmic treatment was prescribed.

Clinical signs had been well-controlled for 4 months following the diagnosis of ARVC, when acutely decompensated congestive right heart failure led to euthanasia. This survival time is greater than previously reported for canine ARVC, and although purely speculative, L-carnitine and/or taurine supplementation may have exerted a beneficial effect in this regard.

Arrhythmogenic right ventricular cardiomyopathy is histologically characterised by progressive fibro-adipose replacement of right ventricular myocardium in humans[6,8–10] and dogs[5,23,25]. However, these changes are not specific for a diagnosis of ARVC[9], and negative endomyocardial biopsies also do not exclude the condition[3,9,21]. Myocardial histology could unfortunately not be performed in the dog in this report. However, cardiac histology is not a prerequisite for the diagnosis of ARVC in humans[6], and the echocardiographic diagnosis of the condition has been well-established[20].

It is believed that a diagnosis of ARVC is justified in this dog owing to the similarities with humans, dogs and cats suffering from the condition, as regards patient age, clinical signs, ECG, radiographic and echocardiographic findings, and the exclusion of other causes of isolated right ventricular failure. Arrhythmogenic right ventricular cardiomyopathy should be considered a differential diagnosis in any young dog presented with isolated right-sided cardiac failure[23,25], syncope[5], or unexplained ventricular tachyarrhythmias[5].

ACKNOWLEDGEMENTS

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Book review — Boekresensie

An update on zoonotic diseases

Coordinated by P Pastoret

This volume of the *Revue Scientifique et Technique* constitutes another valuable contribution of the Office International des Épizooties (OIE) to global control of animal diseases. As the coordinator has pointed out in the conclusion to the volume, the risk of transmission of diseases from animals to humans is increased by more intensive modern farming practices, globalisation of trade in animal products, and, possibly, global warming, because they upset equilibria. The last decades have seen the emergence of apparently new zoonoses such as new variant Creutzfeldt-Jakob disease, Nipah virus, avian influenza, Ebola virus, and many others. The main focus of the publication is on the newer zoonoses, but an interesting introductory chapter deals with the history of zoonoses. This chapter underlines the problem that, in the absence of accurate knowledge of the aetiology and epidemiology of zoonoses, recommendations for prophylaxis are likely to be wide of the mark.

A chapter on animals and public health emphasises the challenge that zoonotic diseases pose in the already complicated balance that must be achieved between optimum production and globalisation of trade in animal products to ensure food security on one hand and conservation of the environment, biodiversity and animal welfare on the other. The example of cowpox is used to illustrate the ability of potentially pathogenic agents to interact with different hosts in different ways. This theme is expanded in a chapter on the emergence of zoonoses when pathogens cross the species barrier, which in humans is promoted by population growth and mobility. Chapters covering the effects of climatic change on arboviral infections and vectors, and the molecular evolution of viruses, which has resulted in the development of RNA viral 'quasispecies', characterised by considerable genomic variation, contribute to our understanding of the apparent unpredictability of zoonotic outbreaks.

Successive chapters deal more extensively with particular zoonoses: Hantavirus infections, haemorrhagic fevers caused by Bunyarviridae and Filoviridae (in particular Rift Valley fever, Marburg and Ebola virus), monkeypoxvirus, new variant Creutzfeldt-Jakob disease, Lyme disease, cat-scratch disease, Hendra and Nipah virus, West Nile virus, bat lyssavirus, recent developments in influenza virus infections, and Borna disease virus. With the possible exception of the chapter on monkeypoxvirus, which I found disappointing because it was devoted largely to expressing the author's view that the remaining stocks of smallpox virus should be destroyed, the chapters provided in-depth and highly informative overviews of these topical diseases. Two chapters devoted to bacterial food-borne and parasitic food- and waterborne diseases brought one back to the familiar and provided an interesting update on salmonellosis, campylobacteriosis, *Escherichia coli* infections, toxoplasmosis, sarcocystosis, giardiosis, cryptosporidiosis, and infection with various flukes, tapeworms and nematodes. After the preceding chapters, reading about agents that are destroyed by thorough boiling and cooking offers some hope of survival in an increasingly threatening world. A chapter on the possible dangers of xenotransplantation puts the reader firmly back in the present/future, and presents a lucid exposition of the possible dangers, mainly in the domain of potential viral infection, and how such agents might be detected and avoided.

The final chapters deal with the evolution of zoonoses and the measures that may be applied to limit their development, and the public health implications of zoonoses.

This publication is essential reading for all veterinarians who are involved in the control of animal diseases in the interests of public health. It contains valuable information for anybody who has any contact with animals, whether for food production, companionship, or recreation, or simply by accident while indulging in pastimes such as hiking and touring. It is readable and generally well-written, with relatively few editorial lapses: examples are the incorrect use of data as a singular subject in some chapters, and in particular the chapter on Borna disease, which has a strongly non-English flavour and a photograph of mice labelled as 'litter maids'. All except two of the chapters (the history of zoonoses, which is in French, and the chapter on quasispecies, in Spanish) are in English, with French and Spanish summaries. Several of the chapters contain beautifully reproduced and informative illustrations, several in colour. I unreservedly recommend this publication on a subject that impinges increasingly upon the daily lives of human beings everywhere.

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