1st CEVA Cardiology Award
for Interns and Residents

Best Clinical Cases Report
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Ceva Santé Animale is keen to **encourage and motivate European students in the area of veterinary cardiology.** The company has therefore created two prizes which will be awarded each year for the best clinical cases presentations, at Internship and Residency levels respectively.

The two first winners at intern and resident level received their awards in front of the prestigious jury of the award and a room full of cardiology experts during Ceva’s second Human and Veterinary Crosstalk Symposium on Aldosterone, in Bordeaux (France) on October 1st, 2011.

“These students are tomorrow’s specialists, explained Sylvie Bourrelier, Western Europe Marketing Director - Companion Animals for Ceva. They need to acquire a certain scientific methodology. In opening this competition, we want to give an incentive to students (and a tool to their teachers) to prepare a quality case study in English, a skill they need to complete their studies and in their future professional career.”

“This award is perfectly in line with our vision “Together beyond animal health”, added Ceva CEO, Dr. Marc Prikazsky. Through this initiative, we work with teaching professors and students across Europe, as well as research groups from both the human and veterinary health sectors to promote and advance scientific knowledge.”

Each year, the participants from both categories will be given the chance to attend a major European veterinary congress, with flights, accommodation and registration covered by Ceva Santé Animale. They will also have the opportunity to present their case orally or through a poster at a veterinary event, where their tutor will also be invited.

In this booklet, you will find a presentation of this Award. The first part describes the candidate application as well as the jury and the way candidates are assessed. You will also be able to read testimonies given by the participants of the first edition. Their cases are presented at the end of this document.

The organizing committee:

Sylvie Bourrelier  
DVM. Western Europe Marketing Director  
Companion Animals  
Ceva Santé Animale

Emilie Guillot  
DVM. Technical Manager Cardiology  
Companion Animals  
Ceva Santé Animale
The Candidates application

The candidates for this award should be graduates in veterinary medicine carrying out an internship or residency in internal medicine/cardiology within a veterinary school or private practice. Based in the European Community they should be able to present their results in English.

To participate, the first step is the registration at the electronic address Prilactone@ceva.com. Ceva provides the registered applicant with draft power point files and word for support.

The clinical cases presented should involve the diagnosis and management of a case of heart failure in a dog or a cat. All cases of heart disease can be included except congenital cases.

The final applications sent to Ceva, in an electronic format in English should include:

- a short abstract (max 500 words)
- a power point for oral presentation of the case
- a word document suitable for publication containing a description of the diagnostic procedure, follow up of the animal and literature review.

The Jury

The Jury made up of independent international experts in cardiology from veterinary and human medicine has to evaluate the applications in order to select a final panel in both Internship and Residency. The evaluation criteria are based on the originality of each case, the description of diagnostic procedure and follow up (scientific evaluation) and the literature review.

The jury designed a specific evaluation procedure covering the overall organization, the quality and educational value of the case and the scientific and technical quality on both abstract and presentation.

The Jury at the Award presentation during the Ceva Cardiology Symposium that took place in Bordeaux, October 1st 2011.

Rebecca STEPIEN
DVM, MS, DipACVIM (Cardiology)
USA

Nuala SUMMERFIELD
BVM&S, DipACVIM & ECVIM-CA (Cardiology), MRCVS
UK

Faiez ZANNAD
MD, PhD, Research Director
France

Michele BORGARELLI
DVM, PhD, DipECVIM-CA (Cardiology)
USA

Frédéric JAISSER
MD, PhD, Research Director
France

Mark OYAMA
DVM, DipACVIM (Cardiology)
USA
The 2011 Ceva European students cardiology award

17 applicants, 4 countries
- Pre-selection Internship Award: 6 cases from 4 countries (UK – FR – SW – IT)
- Pre-selection Residency Award: 4 cases from 3 countries (UK - IT – SW)

“We were very pleased for the first edition of the award to receive 10 fully written up cases from 4 countries, commented Sylvie Bourrelier. This is quite satisfying because we were addressing a very narrow target. However we are hoping to have more applicants next year because the award will be better known. We are also delighted that such a prestigious jury accepted to collaborate with Ceva on this award. Based on the concept of the cross talk between human and veterinary specialists, a common evaluation procedure got the members of the jury to share their expertise as professors and agree on the winners. We would like to express our genuine gratitude to each of them for devoting their expertise and time to the project.”

The prizes were awarded during the Ceva Cardiology Symposium on October 1st 2011, a Ceva event organized every two years. Travel costs and accommodation for the winners to attend this symposium were offered by Ceva. The tutors of the winning entries were also invited to the Ceva Cardiology Symposium.

The 2011 Resident Awarded received the registration to the ECVIM Congress in Maastricht in September 2012 (Congress of the European College of Veterinary Internal Medicine – Companion animals), with flight tickets and accommodation for the duration of the congress.

The 2011 Internship Awarded received the registration to the SEVC (Southern European Veterinarian Conference) in Barcelona, including flight tickets and accommodation for the duration of the congress.

Testimonials from the tutors

“This award is very good thing because it challenges the students. They are required to be familiar with this approach during their fifth year as they have to present one of their clinical cases as a case study. When students take part in this kind of competition, they come and tell us about their case, they are given general ideas and advice, but we do not correct them. It must remain their own work to be evaluated by the jury. Being assessed and recognized by a panel of experts from both Human and Veterinary medicine is really motivating as well as attending a major international conference .”

Armelle Diquélou
Dr Vet, PhD, Assistant Professor, Internal Medicine, National Veterinary school of Toulouse (France) - Tutor of Sandra Contassot, winner of the Internship award.

“I was proud to have Jordi win, and Brigite short-listed for the CEVA awards. There is not a similar prize in veterinary cardiology. Winning such a prize brings a significant esteem factor, which the winners and shortlisted candidates can include on their CVs .So, they should really gain from this.”

Dr Joanna Dukes-McEwan
BVMS (Hons), MVM, PhD, DVC, DipECVIM-CA (Cardiology), MRCVS-Tutor of Jordi Lopez Alvarez, winner of the Residency award
RESIDENCY CASE STUDY

Jordi López-Alvarez

LitVet, MRCVS
Resident ECVIM (Cardiology),
April 2008 - May 2011
University of Liverpool, Small Animal Teaching
Hospital Leahurst, Chester High Road, Neston, UK
jlopezalvarez@rvc.ac.uk

Jordi graduated as Licenciat in Veterinary Medicine from the Universitat Autònoma de Barcelona (Spain) in 2003. During his studies, he spent 2 years in the École Nationale Vétérinaire d’Alfort, Paris (France) and worked in first opinion practice there, before going back to Spain. He then completed an 18-months internship at the Hospital Clínico Veterinario (HCV-UAB) in Barcelona, finishing in October 2004. After three and a half years working in private practice in Barcelona he moved to Liverpool to take the position as Resident in small animal Cardiology. Jordi is currently working towards his PhD about the natural history of mitral valve disease in dogs, at the Royal Veterinary College in London.

Jordi was given the opportunity to give an oral presentation of his case during Ceva’s second Human and Veterinary Crosstalk Symposium that took place in Bordeaux on October 2011.

“When I first saw the e-mail inviting me to participate to the Ceva residents’ case report competition, I thought it was a fun thing to get involved with. In my opinion, competitions are always challenging and interesting because they stimulate you to do your best and give you the opportunity to have social interaction with other cardiology residents in Europe. The competition was especially interesting because of the prize, which was double. I was invited to an excellent state-of-the-art scientific meeting in Bordeaux and yet to come, Ceva is taking me to the ECVIM-CA congress in 2012 in Maastricht. I would encourage all the interns and residents to participate in future competitions as it is good fun and rewarding.”
CHASING FLUID AWAY! - TREATING THE PROGRESSIVE CONGESTIVE HEART FAILURE PATIENT.

CASE STUDY OF MYXOMATOUS DEGENERATIVE VALVULAR DISEASE IN A GERMAN SHEPHERD DOG

Jordi López-Alvarez a, b

Abstract
Myxomatous degenerative valvular disease (MDVD) is the most common acquired cardiac disease in small breed dogs, characterized by slow and progressive degenerative thickening of the valvular leaflets, frequent rupture of minor order chordae tendinae and secondary valvular prolapse. The loss of leaflet coaptation generates a regurgitant orifice that results in the regurgitation volume flowing backwards into the atrium in each ventricular systole, causing progressive volume overload, increased diastolic filling pressures and eventual development of congestive heart failure. German Shepherd dogs (GSD) show less florid mitral valve thickening and the main cause of their valvular incompetence may be primary valvular prolapse. In large breeds compared with small breed dogs, MDVD has faster progression with worse hemodynamic consequences, leading frequently to myocardial failure, arrhythmias and poor outcomes. It is very important to consider this valvular cause of volume overload in large breed dogs to avoid misdiagnosing idiopathic dilated cardiomyopathy, as both conditions share similarities in their clinical presentation.

Treating congestive heart failure (CHF) is palliative. The underlying disease will continue to progress and different strategies to control edema, effusions and clinical signs are required. Different diuretic classes to perform sequential nephron blockade, up-titration of heart rate medication, afterload reduction and improvement of abdominal discomfort are some of the available options. This case, a 4 years old GSD with MDVD, atrial fibrillation, myocardial failure, post-capillary pulmonary hypertension and CHF, demonstrates this approach.

Running header: MDVD in a GSD
Keywords: Congestive heart failure, diuretics, sequential nephron blockade, atrial fibrillation, torasemide.

a Small Animal Teaching Hospital, University of Liverpool, School of Veterinary Science, Leahurst, Chester High Road, Neston, CH64 7TE, UK
b Present address: The Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, Herts AL9 7TA, United Kingdom.

Case
A 4 years old, neutered male German Shepherd dog (GSD) was referred for progressive exercise intolerance and tachypnoea over the previous month. No history of previous illnesses was reported and he was fully wormed and vaccinated.

On physical examination, the dog was bright, alert and responsive and the body condition was adequate. He was tachypnoeic (50 rpm) and presented marked abdominal distension with positive fluid thrill and hepatojugular reflux. The mucous membranes were pink and moist, with normal capillary refill time. He presented irregularly irregular tachycardia (240 bpm) with variable pulse volume and significant pulse deficits (pulse rate <100/minute). Auscultation of the chest revealed a variable grade II-III/VI left apical systolic heart murmur, and no adventitious but harsh bronchovesicular lung sounds. The body temperature was normal at 38.4°C.

Systolic blood pressure, indirectly assessed with a Doppler device, was adequate at 150 mmHg. Hematology and biochemistry profile were within normal ranges, showing only mild elevation of liver enzymes. Severe cardiomyocyte damage was confirmed with cTnI measurement of 1.18 ng/mL (ref val. <0.15). Six lead electrocardiogram was diagnostic of atrial fibrillation, and presented ST segment coving with border-line high QRS complex duration, considered a non-specific sign of left ventricular enlargement/hypertrophy and myocardial hypoxia (Figure 1).

Abdominocentesis revealed the presence of a modified transudate. Doppler echocardiography showed subjectively mild thickening and prolapse of the mitral valve leaflets, severely hypokinetic and dilated left ventricle and severe left atrium and pulmonary venous dilation. Color Doppler showed moderate to severe mitral regurgitation (Figure 2). Mild tricuspid regurgitation with slightly elevated velocities was suggestive of mild post-capillary pulmonary hypertension. The left ventricular systolic function was severely impaired. The diastolic left ventricular filling pressures were estimated to be elevated, suggestive of left sided congestive heart failure (CHF). Mild pleural and pericardial effusions were present,
consistent with right sided CHF. The final diagnosis was myxomatous degenerative valvular disease (MDVD) with atrial fibrillation, myocardial failure, mild pulmonary hyper-tension and biventricular CHF.

The dog was admitted to the Hospital for stabilization. A cephalic catheter was placed and intravenous furosemide (1 mg/kg) administered hourly until improvement of the respiratory clinical signs was detected. Adjunctive therapy for CHF at this stage consisted of topical nitroglycerine 2% unguent applied every 4 hours, pimobendan (0.3 mg/kg twice daily) and spironolactone (2 mg/kg once daily). The dog was started on digoxin (3 µg/kg twice daily) and diltiazem (2 mg/kg three times daily) for heart rate control. Once the respiratory rate reduced to <40 rpm, nitroglycerine was stopped and furosemide reduced to four times daily and subsequently changed to oral furosemide tablets (2 mg/kg three times daily). At this stage the heart rate was stable at 140-150 bpm, and improved peripheral pulses (<130/min), reduced body weight and decreased abdominal distension were noted.

**ECG report:**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Presentation</th>
<th>Reference values</th>
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<tbody>
<tr>
<td>Heart rate</td>
<td>220 bpm</td>
<td>70-160 bpm</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Irregularly irregular</td>
<td>SR/SA</td>
</tr>
<tr>
<td>P wave</td>
<td>Absent</td>
<td>&lt;0.4 mV x 0.04 s</td>
</tr>
<tr>
<td>P-R interval</td>
<td>n/a</td>
<td>0.06 – 0.13 s</td>
</tr>
<tr>
<td>R wave height</td>
<td>2.8 mV</td>
<td>&lt;3.0 mV</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.06 s</td>
<td>&lt;0.06 s</td>
</tr>
<tr>
<td>Q-T interval</td>
<td>0.16 s</td>
<td>0.15 – 0.25 s</td>
</tr>
<tr>
<td>ST Segment</td>
<td>Coving</td>
<td>Not elevated or depressed</td>
</tr>
<tr>
<td>T wave</td>
<td>&lt;25%, negative</td>
<td>&lt;25% height R wave</td>
</tr>
<tr>
<td>MEA</td>
<td>+80°</td>
<td>+40° – +100°</td>
</tr>
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</table>

**Fig. 1:** ECG at presentation: standard six lead ECG diagnostic of atrial fibrillation, at the origin of the irregular tachycardia with pulse deficits detected on physical examination. Notice ST segment coving with border-line QRS complex duration, considered unspecific signs of LV enlargement/hypertrophy and myocardial hypoxia. Leads I, II and III, paper speed 50 mm/s, sensitivity 1 mV= 1 cm. Six lead ECG recorded with the patient in right lateral recumbency.

**Fig. 2:** Doppler echocardiography at presentation. End diastolic (A) and end-systolic (B) frames obtained from the right parasternal, long axis, 4 chamber view; these images show a rounded, volume overloaded left ventricle with nodular thickening of the atrioventricular valves and prolapse. (C) Left ventricular M-mode obtained from the right parasternal, short axis view at the base of the papillary muscles; this image show reduced radial motion. (D) Doppler color map of the left atrium in mid systole obtained from the left apical, 4 chamber view; this image show moderate to severe MR.
Thoracic radiographs revealed cardiomegaly (predominantly left sided), generalized mild interstitial lung pattern and mildly congested lobar veins, all suggestive of residual left sided CHF (Figure 3). Biochemistry profile showed mild azotemia (iatrogenic pre-renal) and benazepril treatment was initiated (0.5 mg/kg once daily). The dog was discharged three days later with oral medication.

The owner was instructed to monitor signs of progression at home, assessing body weight, respiratory and heart rates, which were also recorded at each revisit. An initial Holter monitor was performed to assess ventricular rate control at home in order to adjust the antiarrhythmic medication. Biochemistry profile, mainly to assess kidney function and electrolytes, was monitored after each new diuretic introduction or increase of their doses. cTnI was measured to assess severity of disease and as prognostic indicator. Digoxin dose was optimized to achieve trough levels between 0.5 and 0.9 ng/mL. The consequent follow-ups were planned bearing this scheme in mind (see Figure 4).

Fig. 4: Graph representing the progression of the body weight and the different actions taken during the follow up of the case. The blue arrows represent cTnI measurement, the green arrows trough digoxin seric levels and the thick red arrows abdominocentesis (with the amount of abdominal fluid retrieved in litres). The capital letters indicate treatment modifications. The purple * symbol represents 24 hours Holter monitor recordings.
Discussion

Myxomatous degenerative valvular disease is the most common cardiac disease in small breed dogs. It is a relatively benign condition in these breeds, characterized by an unpredictable but usually slowly progressive degeneration of the valvular apparatus. Histologically, this process is characterized by an absence of inflammatory infiltrates, proliferation of valvular interstitial and endothelial cells, abnormal deposition of extracellular matrix and disorganization of the collagen network. These structural changes are responsible for the typical disrupted macroscopic aspect of the affected valves, with thickening of the leaflets, loss of coaptation and chordae tendinae rupture. This valvular incompetence generates hemodynamic consequences consistent with volume overload and increased filling pressures that can eventually lead to CHF. Large breed dogs also suffer MDVD, but the progression seems to be much faster and more severe for these breeds, with less valvular structural changes but worse hemodynamic consequences, leading frequently to myocardial dysfunction, arrhythmias and poor outcomes. Valvular prolapse rather than chronic degeneration of the leaflets might be the cause of their valvular incompetence. This is a relatively subtle change that requires careful evaluation by an experienced and observant echocardiographist for its diagnosis. The resulting volume overload increases preload, stimulating contraction by the Frank-Starling mechanism, and the mitral regurgitation reduces afterload. Increased preload and reduced afterload results in hyperdynamic ventricular contraction and reduced peak systolic wall stress. This stimulates replication of sarcomeres in series (eccentric hypertrophy), but this mechanism is still insufficient to normalize the increased diastolic wall stress, and might explain why large breed dogs develop myocardial systolic dysfunction more prematurely and acutely than small breeds.

Dilated cardiomyopathy is a disease of the cardiac muscle characterized by reduced systolic function. It manifests as progressive dilation of the cardiac chambers due to volume overload, which frequently leads to supraventricular arrhythmias, namely atrial fibrillation. Familial predisposition has been suspected in large breed dogs for a long time, and in recent years increasing evidence points towards a genetic origin, with a few genetic mutations already published. However, until more accurate genetic tests are available, its diagnosis still needs to be done by exclusion of diseases sharing similar clinical presentation. Neither a genetic basis nor a familial predisposition for DCM has been suspected in GSDs, and furthermore some studies found that DCM is a rare condition for this breed, suggesting that exquisite attention is required to avoid misdiagnosis.

Heart failure is a clinical syndrome characterized by the inability of the heart pump to make blood advance, and CHF is the result of backwards accumulation of fluid and the neuro-hormonal compensatory mechanisms activated. Due to the distribution of the vascular system, detection of CHF from the right side of the heart is performed by clinical examination (eg: distension of the jugular veins, positive hepatojugular reflux, ascitis). Left sided CHF can be first suspected by clinical examination and auscultation, but the definitive assessment of the severity of the condition relies on radiology or echocardiography. It has now been shown that the diastolic filling pressures of the left ventricle can be estimated by Doppler echocardiography, measuring the transmural inflow pattern velocities (E and A waves) and the duration of the isovolumic relaxation time (IVRT). Those parameters are dependent on loading conditions and heart rate, but the ratio E:IVRT is not, and seems to be very accurate for the detection of CHF. We used this echocardiographic measurement to monitor left sided CHF in this case, which was very practical, as it limited the amount of thoracic radiographs, which due to the presence of pleural effusion, would not have always been easy to interpret.

Applying the Bernouilli equation on the systolic tricuspid regurgitation and the diastolic pulmonic insufficiency velocities gives a good indication of the pulmonary circulation pressures and allows them to be followed up over time. This is another interesting application of echocardiography, which helped in the assessment of this case, as it negates the need for invasive right sided cardiac catheterization. Pulmonary hypertension can be classified as either pre-capillary, when primary lung pathology increases arterial resistance, or post-capillary, when there is increased pressures in the left atrium that increase the pressure in the pulmonary circulation. Post-capillary hypertension is recognized in 14-31% of cases of chronic left sided cardiac dysfunction in dogs. This is best treated by reducing the left atrial pressures, but in some advanced cases this is deemed to be difficult and adding arterial vasodilators selective for pulmonary circulation (ie: phosphodiesterase V inhibitors) can be beneficial. In this case, sildenafi l was recommended, but due to economic reasons the owner decided against it.

In the presence of CHF, several compensatory neuro-hormonal mechanisms are activated in response to low cardiac output. Those alleviate the clinical signs in first instance, but contribute with the progression of the disease when chronically activated inducing vasoconstriction, edema and increased blood volume. These mechanisms include: sympathetic system activation, with vasoconstriction and positive chronotropic and inotropic cardiac effects; renin-angiotensin-aldosterone system (RAAS) activation, with sodium and water retention, cardiac remodeling and vasoconstriction; secretion of vasopressin promoting dilutional hyponatremia (correlating with adverse outcome in CHF) may aggravate cardiac remodeling and peripheral vascular resistance; release of endothelin-1 in response to shear stress, hypoxia, angiotensin and vasopressin eliciting vasoconstriction contributing to systemic and pulmonary hypertension amongst others. The treatment goal for CHF is triple: alleviate clinical signs, stop progression of the disease and prevent complications. To achieve this goals is required the use of diuretics, inotropic support and RAAS blockers. High dose diuretic monotherapy often yields inadequate natriuretic response and resistance (distal nephron hypertrophy, RAAS...
activation, reduced renal flow, decreased GI absorption) and combination of different diuretic classes, named sequential nephron blockade, is recommended to avoid this resistance. Spironolactone is a weak diuretic and does not significantly increases urine production, but it might potentiate the effect of other diuretics. It has been shown that, probably due to its anti-aldosterone effects, it confers improved survival. Torasemide is a loop diuretic with longer action duration than furosemide, adjunctive anti-aldosterone effects, better GI absorption and it has been associated with reduced mortality in people. There are just a couple of studies in veterinary medicine supporting its use, but in this case it resulted in marked improvement of the clinical signs.

Atrial fibrillation is the more common supraventricular arrhythmia in large dogs in part because of their bigger atrial mass. It is caused by multifocal ectopic triggers with reentrant waves and enhanced by fibrosis, inflammation and wall stretch. Atrial fibrillation suppresses around 20% of the atrioventricular filling by preventing atrial contraction, which can precipitate or worsen CHF, and also can induce myocardial failure due to the fast heart rate (tachycardiomyopathy). Rate rather than rhythm control is the favorite strategy in the presence of structural heart changes, and this is best achieved combining digoxin and diltiazem. Digoxin dose was optimized to achieve trough levels between 0.5 and 0.9 ng/mL, which is lower than the laboratory reference value, but it has been shown to have optimal parasympathetic effect and reduced toxicity compared with higher levels.

Treatment of chronic cases of MDVD can be somehow frustrating, but knowledge of the pathophysiological mechanisms involved and anticipation of possible complications can alleviate clinical signs and offer an adequate quality of life to our patients.

**Abbreviations.**

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<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>bpm</td>
<td>Beats per minute</td>
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<tr>
<td>CHF</td>
<td>Congestive heart failure</td>
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<tr>
<td>cTnl</td>
<td>Cardiac troponin I</td>
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<tr>
<td>DCM</td>
<td>Dilated cardiomyopathy</td>
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<tr>
<td>GSD</td>
<td>German Shepherd dog</td>
</tr>
<tr>
<td>MDVD</td>
<td>Myxomatous degenerative valvular disease</td>
</tr>
<tr>
<td>RAAS</td>
<td>Renin-angiotensin-aldosterone system</td>
</tr>
<tr>
<td>rpm</td>
<td>Respirations per minute</td>
</tr>
<tr>
<td>SR</td>
<td>Sinus Rhythm</td>
</tr>
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<td>SA</td>
<td>Sinus Arhythmia</td>
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**References.**

INTERNERSHIP CASE STUDY

SANDRA CONTASSOT

DVM, Intern student, 2009-2010, National Veterinary School of Toulouse, FRANCE
Emergency and Critical Care Unit, 2010-2011, National Veterinary School of Toulouse, FRANCE

Sandra graduated from the National Veterinary School of Lyon in 2008 and followed an Internship in the National Veterinary School of Toulouse in 2009. She worked as a Clinical Assistant in the Toulouse Emergency and Critical Care Unit in 2010. Sandra is currently working in a veterinary practice in La Réunion.

Sandra's poster of her case study was presented during Ceva’s 2nd Human and Veterinary Crosstalk Symposium.

“Cardiology has always been my favorite medical subject, my thesis treated valvular disease in dogs and the idea to participate in a competition was very motivating. Such a competition allows the European students to confront and gives the opportunity to exchange with a tutor in a privileged relationship that would not necessarily exist in the context of the internship. To work on a clinical case also allows to get acquainted with the diagnostic and scientific approach, and obliges us to make the bibliography to better understand the mechanisms involved in cardiac disease. I was very happy to proudly represent the school of Toulouse in a European competition.”

TUTOR

Armelle Diquélou

DVM, MSc, PhD, Assistant Professor, Internal Medicine, National Veterinary School of Toulouse, France
a.diquelou@envt.fr
A CLINICAL CASE OF DILATED CARDIOPATHY IN A CAT

Sandra Contassot*, Thomas Daste#, Armelle Diquélou*

Introduction

Feline cardiomyopathies are divided into different forms, primarily based on echocardiographic criteria. The most common form is idiopathic hypertrophic cardiomyopathy (HCM), characterised by ventricular concentric hypertrophy without identification of a primary cause. The second one is restrictive cardiomyopathy, a myocardial disease affecting the left ventricular diastolic function, leading to left atrial dilation. The third form is dilated cardiomyopathy (DCM) consisting of a primary systolic myocardial failure with dilated left ventricular cavity. Since the recognition of the taurine deficiency-induced DCM in 1992 and the further dietary taurine supplementation, the prevalence of feline DCM has decreased and is nowadays about 10% of all feline cardiomyopathies. This report describes the clinical presentation of a cat with decompensated heart failure due to a cardiopathy presumed to be DCM. However, end stage evolution of previous undiagnosed heart diseases could not be excluded. After emergency stabilization, the response to long term treatment (diuretics, pimobendan and ACEI) was quite good and the cat was still alive 6 months after first presentation. This emphasizes the difficulty of accurate diagnosis and prognosis of feline cardiomyopathies.

Case history

A 12-year-old female neutered domestic shorthair cat was presented to the Intensive Care Unit of the National Veterinary School of Toulouse for severe respiratory distress (Fig. 1). The owners had observed a mild dyspnoea for 4-6 weeks and a decreased body condition for one year with dysorexia, lethargy and exercise intolerance. The cat, not vaccinated nor dewormed, was fed a commercial feline maintenance diet.

On physical examination, the cat showed hypothermia (36.4°C), tachypnea (respiratory rate 58 breaths/min) and respiratory distress with extended neck. Thoracic auscultation revealed muffled lung sounds, bradycardia (heart rate [HR] 140 beats/min) and sternal holosystolic murmur (grade II/VI). The mucous membranes were pale and the femoral pulses were very weak. Abdominal palpation revealed hepatomegaly.

The cat immediately received oxygen supplementation and mild intravenous sedation with 0.3 mg/kg butorphanol (DOLOREX®, Intervet Schering Plough). A thoracic focused assessment with sonography in trauma exam (T-FAST exam) revealed severe pleural effusion without pericardial fluid. Bilateral thoracocentesis was performed and approximately 150 mL of sero-hemorrhagic fluid was obtained (Fig 2). Following thoracocentesis, breathing was improved and further examinations were possible.

The pleural fluid analysis revealed a modified transudate. Thoracic radiography confirmed the severe pleural effusion despite the previous thoracocentesis and revealed pulmonary edema (Fig. 3). Heart size and cardiac shape could not be evaluated. Abdominal radiography confirmed the slight hepatomegaly (Fig. 4). Electrocardiography showed sinus rhythm. A complete blood cell count (CBC) and plasma biochemical profile were performed. No significant changes were noted on the CBC but the biochemical panel revealed mild hyponatremia (144 mmol/L ; reference interval (RI) [148 – 157] mmol/L), mild increase in creatinine concentration (183 µmol/L ; RI [80- 180 µmol/L]) and elevated alanine aminotransferase activity (ALT) (806 U/L ; RI, [20 - 107 U/L]). Tests for FeLV, FIV, heartworms, hyperthyroidism and taurine deficiency were considered but could not be performed for financial reason.

* Internal Medicine, INP-Ecole Nationale Vétérinaire de Toulouse, 23 chemin des Capelles, 31 076 Toulouse cedex 3, France
# Emergency and Critical Care, INP-Ecole Nationale Vétérinaire de Toulouse, 23 chemin des Capelles, 31 076 Toulouse cedex 3, France
Echocardiography revealed markedly decreased fractional shortening (FS) and increased left ventricular diastolic and end-systolic internal diameter (LVID) (table 1, Fig. 5A), increased E point to septal separation (E-Se) (table 1, Fig. 5B) as well as an increased left ventricular pre-ejection period (PEP) to ejection time ratio (PEP/LVET) (table 1, Fig. 5C). The left atrium was mildly dilated (left atrium /aortic root ratio 1.7, usual value < 1.5) with a mild mitral valvular insufficiency (Fig. 5D).

A diagnosis of dilated cardiopathy with severe cardiac systolic dysfunction and mild renal failure was made. During the first 24 hours, the cat needed a second thoracocentesis; it received intravenous furosemide 2 mg/kg q 6h (DIMAZON®, Intervet Schering Plough) and pimobendan 0.25 mg/kg q 12h per os (VETMEDIN®, Boehringer Ingelheim). After clinical improvement, furosemide was decreased to 1 mg/kg q 12h per os, pimobendan was maintained [0.25 mg/kg q 12h per os] and benazepril was added (0.5 mg/kg q 24h per os) (FORTEKOR®, Novartis). At discharge, 3 days post admission, the cat was eupneic, with a heart rate (HR) of 180 beats/min, but still had pale mucous membranes and weak femoral pulses.

Table 1. Echocardiographic values in the cat obtained (in duplicate), for ventricular values, on TM echocardiograms from both the right short and long axis left ventricular views; for left atrium (LA) and aortic root (Ao), on the bidimensional short axis early diastolic aortic view, for E-Se on right long axis mitral TM echocardiograms, for PEP and LVET on right 5 chambers TM echocardiograms, and for mitral valve regurgitation (MVR) on spectral continuous Doppler tracing from left parasternal apical 4 chambers view. IVS = Interventricular Septum in diastole (IVSd) and systole (IVSs). LVID = Left Ventricular Internal Diameter in diastole (LVIDd) and systole (LVIDs). LVW = left ventricular wall in diastole (LVWd) and systole (LVWs). Usual values from Sisson et al. J Vet Intern Med (1991), 5 : 232.23 and Atkins et al. J Vet Intern Med (1992), 6 : 55-63

<table>
<thead>
<tr>
<th>Variable</th>
<th>IVSd (mm)</th>
<th>LVIDd (mm)</th>
<th>LVWd (mm)</th>
<th>IVSs (mm)</th>
<th>LVIDs (mm)</th>
<th>LVWs (mm)</th>
<th>SF (%)</th>
<th>Ao (mm)</th>
<th>LA (mm)</th>
<th>LA/Ao</th>
<th>E-Se (mm)</th>
<th>PEP (ms)</th>
<th>LVET (ms)</th>
<th>PEP/LVET</th>
<th>MVR (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed Value</td>
<td>3.8</td>
<td>20.2</td>
<td>3.6</td>
<td>3.6</td>
<td>17.8</td>
<td>4.7</td>
<td>12</td>
<td>8.1</td>
<td>13.6</td>
<td>1.7</td>
<td>7.5</td>
<td>75</td>
<td>117</td>
<td>0.7</td>
<td>1.89</td>
</tr>
<tr>
<td>Usual values</td>
<td>4.2 ± 0.7</td>
<td>15 ± 2</td>
<td>4.1 ± 0.7</td>
<td>6.7 ± 1.2</td>
<td>7.2 ± 1.5</td>
<td>6.8 ± 1.1</td>
<td>52 ± 7</td>
<td>9.5 ± 1.4</td>
<td>11.7 ± 1.7</td>
<td>1.25 ± 0.18</td>
<td>0-2</td>
<td>45 ± 6</td>
<td>116 ± 19</td>
<td>0.40 ± 0.05</td>
<td>None</td>
</tr>
</tbody>
</table>

Fig. 3: Right lateral view of thoracic radiography showing pleural effusion and pulmonary oedema.

Fig. 4: Right lateral view of abdominal radiography showing no abnormality.

Fig. 5A: Right short axis TM echocardiogram at the level of papillary muscles showing dilated left ventricle and decreased shortening fraction.

Fig. 5B: Right long axis TM echocardiogram at the mitral level showing increased E to Septal separation.
Clinical improvement with no dyspnoea but persistent exercise intolerance was observed on control examination, 15 days after discharge. On thoracic auscultation, soft pulmonary crackles and a gallop heart sound were heard in addition to the soft sternal holosystolic murmur. Electrocardiography revealed sinus tachycardia associated with rare ventricular premature complexes. Thoracic radiography showed little pleural effusion but a persistent pulmonary edema. Echocardiography revealed no real improvement of echocardiographic values. Potassium and sodium were within reference intervals but the plasma creatinine concentration was still increased [199 µmol/L]. The response to treatment was considered quite good and the same treatments were continued. For financial reason, owners refused regular control examinations. Six month after first examination, the cat was still alive but it was lost for follow up after that period.

**Discussion**

Based on echocardiographic features, a dilated cardiopathy was diagnosed, presumably a DCM. Due to the lack of medical history and follow up, exclusion of other causes of secondary myocardial failure was difficult.² ³ ⁵ Taurine deficiency and congenital cardiopathies were considered unlikely because of the history, as well as tachycardiomyopathy because of the HR at admission and the persistence of systolic dysfunction after HR normalization. However, volume overload leading to secondary myocardial failure (i.e., severe mitral insufficiency) or end stage HCM⁶ ⁷ could not be excluded. Though the exact nature of the cardiopathy was not determined, it did not influence the therapy.

To alleviate respiratory distress, thoracocentesis was immediately performed. High dose diuretics were then initiated intravenously and continued orally after clinical stabilization.⁸ Pimobendan, even if off-label in cats, was chosen for its positive inotropic property, minor adverse side-effects and rapid effect demonstrated in dogs. It has recently been reported to improve cardiac output and systolic dysfunction in feline DCM.⁹ Beta blockers were not considered because of the advanced heart failure, bradycardia and decreased systolic function.¹⁰ An angiotensin converting enzyme inhibitor (ACEI) was added to decrease congestive signs, reduce myocardial oxygen consumption and limit heart remodelling; however because of the ACEI induced-hypotension, benazepril was prescribed after emergency management and clinical stabilization.

Prognosis of either DCM or end stage HCM is very poor² ³ ⁷ with a median survival time of 11 days.³ However, despite the persistent pulmonary oedema observed at day 15, the cat was still alive 6 months after initial admission, with a preserved quality of life according to its owners. Such a long survival time had been observed in 1/8 cats with DCM by Ferasin et al.³

**Bibliography**

Ceva Cardiology Award for Interns and Residents

*Ceva Santé Animale encourages and motivates European students in the area of veterinary cardiology. The company created a Cardiology Award with two prizes for the best clinical cases presentations, at Internship and Residency levels. The goal of this Award is to develop Interns and Residents scientific methodology and ability to prepare a professional qualitative case study.*