Case Log Number 28

Cardiology

January 12, 2008

'Sarge' Williams Canine 7y M/I Boxer

Sarge was presented to the emergency service at Garden State Veterinary Specialists on January 7th. 2008. His owner was concerned with the increased respiratory effort that Sarge was having as well as some collapse episodes that had occurred at home. Sarge had previously visited his regular veterinarian who had begun a regimen of Phenobarbital on the assumption that the collapse episodes were possibly due to seizure activity.

Upon presentation, Sarge's respiration rate was 24 breaths per minute with slightly increased effort. His heart rate was 100bpm and a quick lead II ECG showed a normal sinus rhythm. His temperature was 101.1F and he was quiet, alert and responsive. He had a known history of significant heart disease, however today there was no obvious murmur or arrhythmias upon auscultation. When palpated, his abdomen had no significant findings other than he was still intact and had an enlarged prostate. He was ambulatory on all four legs with a body condition score of 5/9. Neurologically, Sarge did not seem to have any deficits.

Due to Sarge's known history of congestive heart failure (CHF), Dilated Cardiomyopathy (DCM) and Arrhythmogenic Right Ventricular Cardiomyopathy. (ARVC), the emergency clinician was highly suspicious that the seizures suspected by the referring vet may actually be collapse episodes brought on by the significant heart disease. The heart disease had been

medically managed at home by the owner for the past 6months. The medications included Furosemide, Enalapril, Digoxin, Pimobendan, Mexilitine and Sotalol.

With the presenting complaint of collapse and the owner's concern that Sarge may be having seizures, a neurology consult was requested in addition to a cardiology consult. After seeing the neurologist, the Phenobarbital was discontinued after only 6 doses. The possibility of doing an MRI was briefly discussed with Sarge's owner , but not recommended by the neurology service on account of the anesthetic risk due to heart disease and the low diagnostic yield of the test.

Sarge's owner elected to admit him to the hospital for continued close monitoring for one night and then elected to try and take him home the following day. Sarge was admitted to the Intensive Care Unit and an intravenous catheter was placed in his right cephalic vein. Blood was submitted to the lab to check his renal values and electrolytes. This was done to ensure his kidneys were tolerating the diuretics and ACE inhibitors he was getting to control his heart failure. The results were all within normal limits. He was placed on a continuous EKG to monitor his heart rate and rhythm while in the hospital. He continued to stay quiet, alert and responsive and had a wonderful appetite. His medications were continued as they had been previously prescribed. They included: Furosemide 100mg PO every 12 hours, Enalapril 20mg PO every 12 hours, Digoxin 0.1875mg PO every 12 hours, Pimobendan 10mg PO every 12 hours, Mexiletine 200mg PO every 8 hours and Sotalol 80 mg PO every 12 hours. Each medication controls a different symptom of his disease process. Furosemide is a loop diuretic. It is one of the strongest and most effective diuretics used in veterinary medicine to date. It inhibits

sodium and water transport which increases dieresis. With Sarges history of CHF, the resulting fluid buildup in his pleural space would be helped with a diuretic. Enalapril is an Angiotensin – Converting Enzyme (ACE) Inhibitor. The body responds to stressful situations by converting Angiotensin1 to Angiotensin 2. This causes vasoconstriction and other changes resulting in the body essentially being in a constant state of shock. ACE inhibitors cause vasodilatation and decrease aldosterone-induced congestion. Digoxin is an inotropic agent that increases the contractility of the heart as well as reducing the heart rate. Pimobendan is a phosphodiesterase inhibitor that has both vasodilating properties and positive inotropic properties. All of the above drugs were prescribed to Sarge to treat the symptoms of both DCM and CHF. There were also the arrhythmias to consider and treat too. The antiarrhythmics prescribed for Sarge were Mexilitine and Sotalol. Mexilitine is a drug that works like Lidocaine. As a class 1 antiarrhythmic, or sodium channel blocker, it interrupts reentry circuits by slowing conduction and depressing membrane responsiveness. Also, very importantly, it increases the fibrillation threshold in the ventricle. (SmAnCardioMed pg. 509) This makes it an ideal drug for Sarge to try and control his arrhythmias and try to prevent sudden death.

Sotalol is a beta blocker and potassium channel blocker that prolongs the refractory period of the electrical impulse of the heart.

It also may increase the fibrillation threshold in both the atria and ventricle. (SmAnCardioMed pg.514)

During his overnight stay in the ICU, Sarge slept comfortably and had no episodes of collapse. The next morning, Sarge was quiet, alert and responsive. He had a normal temperature of 100.1F His heart rate was 130bpm with good pulses; however, this was after he was taken outside to

urinate. He was pink and his CRT was <2sec. Since he was stable and doing so well, his owner elected to make a cardiology appointment for later in the week and take him home.

Sarge returned 4 days later, on the 12th of January, to be seen by the cardiologist to assess his disease progression. An echocardiogram was performed and showed his left ventricular free wall had limited motion, he had left ventricular volume overload with systolic dysfunction and significant atrial enlargement. There was also significant mitral regurgitation, tricuspid valve regurgitation as well as some pulmonic insufficiency. An electrocardiogram showed a normal sinus rhythm with occasional VPCs. A Holter was recommended to try and evaluate the severity of the VPCs over 24 hours. Holter monitors are helpful tools of the cardiologist to tailor antiarrhythmic medications to each patient. Previous Holters done for Sarge had shown marked improvement of his arrhythmia with medications over the past 1.5 years. Pending the Holter results, Sarge's prognosis remained guarded and his owner was informed of the risk of sudden death.

To clarify Sarges precarious situation, a review of his diagnosis is warranted. Sarge was first diagnosed with DCM, Dilated Cardiomyopathy 1.5 years earlier. DCM is the most commonly acquired heart disease diagnosed in the dog after chronic valve disease (CVD). (VetClinSmAn 34(04)1187-1207) DCM is a primary myocardial disorder characterized by poor contractility and ventricular dilation of the left or both ventricles. Most of the studies for DCM involved Dobermans and Irish wolfhounds, however, Boxers are very close to the top of the list of the most predisposed breeds.

DCM is defined in 3 stages: Stage 1 is defined as having a normal heart on echocardiogram as well as no clinical signs. Stage 2 is defined with an abnormal heart on echocardiogram with no

clinical signs. Stage 3 is defined as having an abnormal heart on echocardiogram as well as clinical signs such as syncope or heart failure. Sarge had already progressed to stage 3 because he had been in heart failure. This was combined with a diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC). This particular diagnosis is also referred to as 'Boxer Cardiomyopathy' because of its prevalence in this breed. ARVC presents in 3 forms. Firstly, it can present as asymptomatic ventricular tachycardia (VT). Secondly, it can present as symptomatic VT, i.e. syncope due to severe VT. Lastly, it can present as right ventricular dilation and severe VT. It is a disease with an adult onset and it can result in sudden death or progress to CHF. Sadly, all too often the first sign of disease is sudden death. Figure 1, shows an M-mode of Sarge's left ventricle. An M-mode, or motion mode, is the real time picture of the systolic and diastolic motion of the myocardium. Figure 2 shows a normal M-mode of the left ventricle. Even just objectively, it is obvious that Sarge has decreased contractility as a result of his disease.

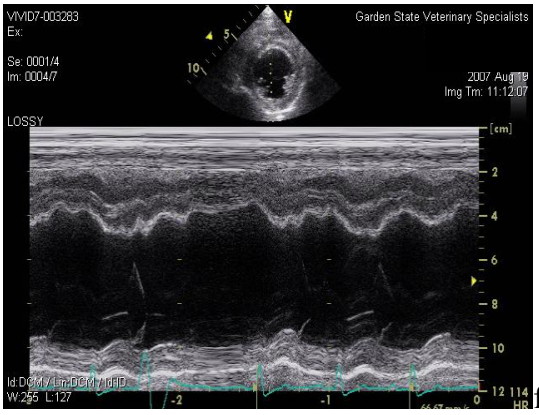

fig.1

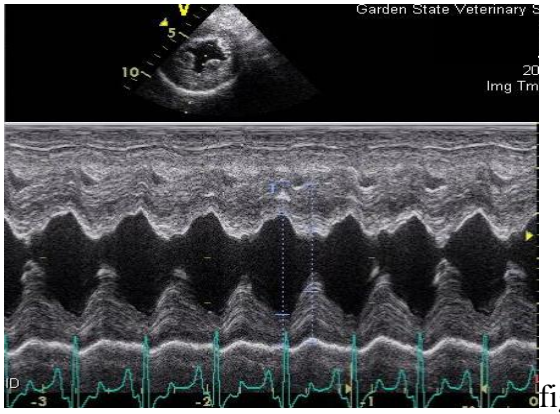
fig 2

Often, the patient presenting with signs of ARVC has an EKG with VPCs with a typical morphology of a Left Bundle Branch Block (LBBB). A Holter monitor is usually recommended

in an ARVC patient in order to have an accurate baseline recording to maximize the titration of the antiarrhythmics to the patient. Through both performing echocardiograms and Holtering, Sarge had been diagnosed with 2 diseases with poor prognosis and he had advanced into CHF.

CHF is defined as having fluid buildup in the lungs or abdomen as a result of decreased heart function. Sarge had progressed to having fluid buildup in his lungs and was therefore being treated with Diuretics (Furosemide) to try and control it.

During this visit, Sarge's owner agreed to another Holter to try and record a syncopal episode. The Holter was applied during his visit and he wore it home for 24 hours. Unfortunately, there were no collapse episodes noted by Sarge's owner. The data showed adequate arrhythmic control at the current treatment he had been receiving. There were 5245 single VPCs, 26 couplets, 3 triplet and no runs of VT. The cardiologist recommended that no medication changes were needed at this time and that a repeat Holter be done in 6 months. This is because, due to the progressive nature of Sarge's disease, the reasons for his suspected syncope would only worsen and therefore need some further medication adjustments.

Sarge continued to do well at home and when his owner called our pharmacy to request refills on his medications, he reported that Sarge seemed to be back to his old rambunctious self.

Then, on the 25th of March, Sarge presented again to the emergency service with some coughing and increased respiratory effort. Sarge had some harsh lungs sounds, so thoracic radiographs were taken and he was given an IV dose of Furosemide. His radiographs showed a mild peribronchial interstitial pattern consistent with CHF. He was admitted to the ICU for observation and IV Furosemide therapy. The Cardiology technician was asked to oversee his care by the ICU and emergency staff. Blood was submitted to the lab to evaluate his kidney

values. The lab results showed a mild azotemia with a BUN of 32 (7-27mg/dl is normal), a creatinine of 2.2 (0.5-1.8mg/dl is normal) and his electrolytes were all within normal limits.

The next day, Sarge seemed bright alert and responsive, His temp was 100.7F and he had not coughed during the night. A repeat of his thoracic radiographs showed resolution of the infiltrates. The two VD thoracic radiographs are pictured below. Figure 3 shows the subtle infiltrates, and figure 4 shows clear lungs fields. Both radiographs show both left atrial enlargement and right side enlargement.

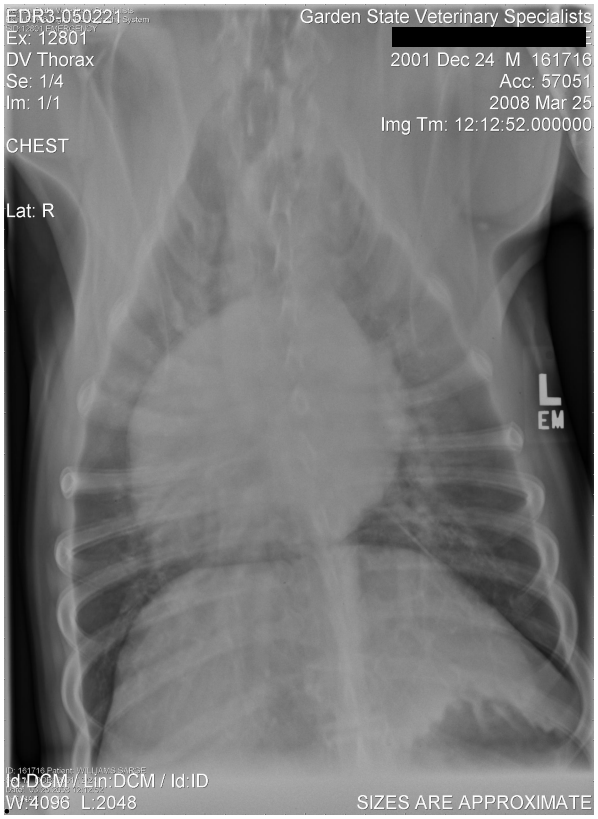

Figure 3.

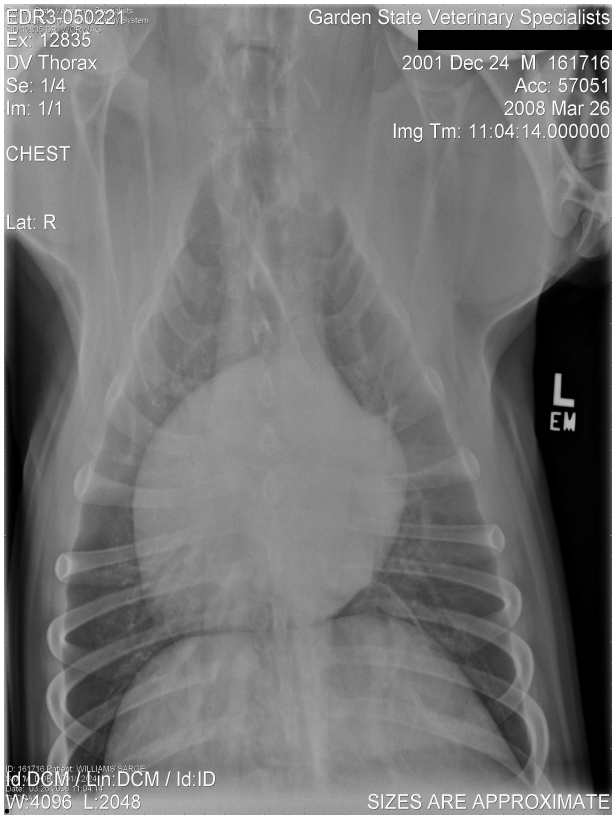

Figure 4.

Sarge was discharged to his owner the 26th of March. Although increasing the dose of furosemide to 100mg TID was considered, the decision was made to keep it at 100mg BID in light of Sarge's azotemia.

His owner was told to keep an eye on his respiration rate and to call if he thought it may be increased. The plan was to have him increase the furosemide at home for a couple of doses and therefore hopefully avoid another trip to the emergency service.

Over the next few weeks, Sarge only returned for bloodwork to evaluate his renal function. His azotemia remained almost static (BUN 42 and creatinine 2.2mg/dl) but his appetite was good at home. Sadly, on April 1st , his owner called concerned that Sarge was not eating well at home. His appetite continued to decline over the next couple of weeks. Then on April 25th, Sarge passed away at home. His owner called and gave us the bad news. He was grateful for the few months of good quality of life we were able to give Sarge. Unfortunatly, Boxer Cardiomyopathy remains a frustrating disease both for owners to deal with and for cardiologist's to treat. It is important that the Cardiology technician communicate with the owners to help them understand what is going on with their pet and how it is being treated. In the case of Sarge, he had 2 disease processess to contend with that both had a poor prognosis. Ongoing research gives us hope that there will be better treatment, and possibly, a way to reverse the disease proccess. Until then we can only do our best to insure the best possible quality of life for our patients and their families.