# Boris the Great
## Canine Dilated Cardiomyopathy

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**Introduction:**

Canines can be affected by a myriad of cardiac diseases that lead to morbidity and mortality.[1] Dilated cardiomyopathy (DCM) is among these disease processes that can cause detrimental effects to multiple body systems in the patient.[3] DCM is considered a primary myocardial disease that causes systolic dysfunction of one or both ventricles and can lead to cardiac enlargement, arrhythmias, and heart failure.[3] There are various causes of DCM such as nutritional deficiencies, idiopathic causes, viruses, Chagas disease, and predisposing hereditary traits.[3]

**History and Presentation:**

Large and giant breed dogs such as Irish Wolfhounds, Great Danes, Doberman Pinschers, and Cocker Spaniels are predisposed to developing DCM.[4] Portuguese water dogs are also predisposed to this disease and typically have a young age onset with high mortality in homozygous recessive puppies.[7] DCM is seen more frequently in male dogs between the ages of four to ten years old.[2] Other risk factors include taurine deficiency, so it is important to evaluate the patient's diet.[2] In the occult phase of DCM, owners may notice clinical signs such as decreased appetite, weight loss, or decreased activity level during the initial onset of this disease.[2] Arrhythmias are not an uncommon finding during this phase and can be an early indicator of DCM.[5] Once in the overt phase, the patient can exhibit clinical signs such as a distended abdomen, tachypnea, anorexia, syncope, or coughing as the disease progresses to congestive heart failure.[5]

A thorough physical examination can reveal tachycardia with the presence of a left sided systolic or right apical murmur, arrhythmia, gallop sound, and/or weak pulses.[6]

Tachycardia commonly occurs as a result of the body attempting to compensate for the decreased contractility of the cardiomyocytes.[6] Due to the thinning of the ventricular walls and subsequent systolic dysfunction, an insufficient amount of blood is ejected from the ventricles into circulation, so the heart rate increases to compensate.[3] If the patient is in congestive heart failure, muffled heart and lung sounds could be appreciated along with jugular pulses.[6] Pulse deficits are commonly associated with patients in atrial fibrillation or patients having premature ventricular contractions.[6] Muscle wasting can also be observed as the disease progresses along with cardiac cachexia.[7]

**Pathophysiology:**

DCM is characterized as the failure of the myocardium to contract normally, resulting in eccentric hypertrophy of one or both ventricles.[3] However, the left ventricle is most commonly affected in this disease process.[3] Studies reveal that DCM in Great Danes is a familial disease with an X-linked mode of inheritance, accounting for the overrepresentation of clinical signs in males.[3] It is believed that females carrying this autosomal recessive trait, although suffering from the disease, do not typically show clinical signs.[3]

Histologically, there are two distinct types of DCM recognized in veterinary medicine.[1] The most prevalent being the attenuated wavy fiber type and the second being fatty infiltration-degenerative type.[1] The attenuated wavy fiber type is considered more prevalent because it can afflict multiple canine breeds.[1] In this type, the myocytes are separated by clear spaces that suggest edematous fluid along with subendocardial fibrosis.[1] The fatty infilitration-degenerative type is considered “Boxer Cardiomyopathy” although it can also be observed in Doberman Pinschers.[1] This type is characterized by

myofiber degeneration and collagen and adipocytes replacing the myocardium.[1] The distinct differences in these two types indicate different disease processes with similar manifestations.[1]

As DCM progresses and contractility is lost, the cardiac output is overall decreased.[5] Therefore, renal blood flow and perfusion is decreased causing the renin-angiotensin-aldosterone system (RAAS) to respond by adjusting sodium and water retention in order to increase the blood volume and venous return to the heart.[9] This increased volume and return causes the affected ventricle to dilate further as it receives the increased hydrostatic pressure that increases the workload of the myocardium.[9] Overstimulation of the myocardium can occur due to the simultaneous release of norepinephrine by the sympathetic nervous system and can result in supraventricular or ventricular arrhythmias, most commonly atrial fibrillation and ventricular premature contractions.[7]

Although compensatory efforts are made by the body, it is impossible to maintain adequate cardiac output for an extended period of time.[2] Eventually congestive heart failure will occur due to the RAAS system promoting fluid retention and increasing the overall cardiac volume on a diseased heart.[3] Since the cardiomyocytes lose their contractility and cannot pump a sufficient amount of blood into circulation the body compensates by increasing the heart rate.[3] However, by increasing the heart rate the diastolic phase of the cardiac cycle is decreased, and the ventricles cannot adequately fill to an appropriate amount.[4] This decrease in filling time further contributes to decreased cardiac output,

continuing a cycle of excessive RAAS system activation and, causing pulmonary edema/effusion, ascites, and respiratory distress.[4]

**Diagnostic Approach/Considerations:**

A thorough physical examination should be performed to assess for any signs of tachycardia, irregular heart rate or rhythm, or congestive heart failure.[6] By using the patient's signalment, history provided by the owner, and clinical presentation a presumptive diagnosis of DCM can be suspected.[6] Further diagnostics such as a minimum database and diagnostic imaging should be pursued for a definitive diagnosis of DCM.[7]

A serum chemistry profile and complete blood count are not necessary for a DCM diagnosis, but can reveal an azotemia, hyponatremia, hypokalemia, and elevated liver enzymes.[7] A stress leukogram may or not be present on evaluation. An electrocardiogram (ECG) will commonly reveal atrial fibrillation or ventricular premature contractions.[3] An ECG is considered a beneficial screening option for dogs in the asymptomatic, or occult, phase of DCM.[7] Holter monitoring is very helpful in early diagnosis of DCM in Doberman Pinschers and Boxers.[7] Studies have shown that the presence of more than fifty ventricular premature contractions, couplets, or triplets over twenty-fours is indicative of future overt DCM, especially in Dobermans.[7]

An echocardiogram is considered the gold standard for diagnosis of DCM.[5] Common findings include ventricular and atrial dilation, thin chamber walls, and a reduced left ventricular fractional shortening and ejection fraction.[5] A normal fractional shortening value ranges from 20-35%, and a normal ejection fraction is greater than 40%.[7] Values outside of these ranges coupled with ventricle or atrial dilation are

definitive for DCM.[9] Doppler evaluation can reveal tricuspid or mitral valve regurgitation due to poor apposition of the leaflets resulting from the chamber dilation.[9] Thoracic radiographs often show a generalized cardiomegaly, pleural effusion or pulmonary edema in more severe cases.[9] In early screenings, left ventricular or left atrial enlargement may be appreciated.[9]

Other diagnostic options include testing for concentrations of the natrieutic peptide (BNP, ANP) and cardiac troponin biomarkers.[7] These biomarkers can be elevated in circulation in cases of congestive heart failure and have been noted in Doberman Pinschers in the occult phase of DCM.[7] Although these biomarkers can aid in diagnosis of DCM, they should be used in conjunction with the aforementioned diagnostics.[7]

**Treatment and Management:**

Treatment can vary depending upon the patient's clinical signs and severity of the disease.[7] In cases diagnosed early without clinical signs, angiotensin converting enzyme (ACE) inhibitors such as enalapril or benazepril should be employed.[3] These medications inhibit the conversion of angiotensin I to angiotensin II, thus promoting vasodilation and decreasing RAAS system activation.[8] Enalapril can be administered at a dose range of 0.25-0.5 mg/kg every twelve hours.[8] Benazepril is commonly administered at 0.5mg/kg every twenty-four hours.[8]

In patients with supraventricular arrhythmias such as atrial fibrillation, digoxin is added to regulate the arrhythmia by slowing the ventricular response rate and increasing contractility of the heart.[3] Digoxin has a narrow therapeutic range and serum levels must be monitored to ensure toxicity is not occurring.[8] Digoxin levels in the serum should be below 1.2ng/L to avoid cardiac and extracardiac toxicities.[8] Clinical signs of digoxin

toxicity include, but are not limited to worsened arrhythmias, decreased appetite, lethargy, vomiting, diarrhea, and behavioral changes.[7] Digoxin should be started within a 2.5-3.0 mcg/kg range and should be adjusted based on how successfully it is controlling the arrhythmia.[8] Diltiazem, a calcium channel blocker, used in conjunction with digoxin yields better results for managing atrial fibrillation and other supraventricular arrhythmias.[7] It works by slowing the AV node and prolonging refractory time.[7]

Pimobendan is a cornerstone therapeutic drug in the treatment of DCM due to its vasodilator and inotropic abilities.[3] This medication inhibits phosphodiesterase III and increases intracellular calcium sensitivity to promote contractility.[3] Pimobendan is considered therapeutic at a dose range of 0.2-0.3mg/kg administered every twelve hours.[8] This medication can be considered in the occult phase of DCM, and is highly recommended in the overt phase, when clinical signs are moderate to severe.[7]

Patients suffering from congestive heart failure or in respiratory distress could require emergency stabilization.[9] A thoracocentesis or pericardiocentesis may be indicated in patients with excess fluid build up in the thoracic cavity or pericardial effusion, respectively.[9] If pulmonary edema or effusions are present, diuretics such as furosemide can be administered at 2-4mg/kg IV initially, then 1-2mg/kg every twelve hours.[8]

Other medical management therapies such as L-carnitine, omega-3 fatty acids, and taurine are available to aid in treatment of DCM.[7] L-carnitine can be used in DCM patients that have low myocardial concentrations.[10] Taurine supplementation is beneficial in patients with low plasma concentrations or patients with a taurine deficiency in their diet.[10]

Although DCM is irreversible, medical management can serve to prolong the quality of life for the patient.[10] Owner compliance is vital in this disease process to ensure the longevity and comfort of the animal.[3] A follow up examination is recommended seven to ten days after initial diagnosis and administration of the medical regimen.[4] A renal panel should be performed to evaluate any abnormalities such as electrolyte imbalances or azotemia due to medications.[4] If digoxin is administered, serum levels must be evaluated in order to avoid toxicities until the proper dosage is achieved.[4] Radiographs can be performed periodically to assess the progression of this disease.[4] Electrocardiograms are beneficial to monitor the presence or control of an arrhythmia, as arrhythmias can lead to sudden death in dogs with DCM.[3] Echocardiograms should be performed every three to six months to evaluate the progression of this disease.[3] Owners must be vigilant and monitor their pet for clinical signs such as labored breathing, lethargy, and collapse as these signs likely indicate disease progression.[3]

**Case Outcome:**

The long term prognosis for DCM is generally guarded to poor, even with medical management.[5] The mean survival time of a patient exhibiting clinical signs is six to nine months.[2] However, studies show that patients not in congestive heart failure can have a mean survival time of up to two years with proper medical and lifestyle management.[2] The onset of DCM in young animals is a negative prognostic indicator.[5] If the patient also has atrial fibrillation, or another type of arrhythmia, the survival time is marginally decreased to three months median survival.[2]

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