Heart Disease in the Irish Wolfhound

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Introduction

Heart disease is unfortunately common in the Irish wolfhound. Valve abnormalities due to endocardiosis are sometimes seen in old wolfhounds, resulting in murmurs, but rarely cause problems. Congenital heart defects occasionally occur in wolfhound puppies but the vast majority of cases which develop heart problems in later life are usually diagnosed as having cardiomyopathy.

"Cardiomyopathy" means "heart muscle disease". The condition occurs in most species of domestic animals and also in humans.

There are a number of different types of this disease, but most cases in the dog are classified as "dilated". Dilated cardiomyopathy (DCM) is characterised by enlargement of all the heart chambers, particularly the main pumping chamber, the left ventricle, thinning of the heart wall and weakening of the force of muscle contraction. This is often accompanied by irregularities of heart rhythm.

Signs of heart failure

Dogs often do not show signs until the function of the heart is severely impaired. This is known as "heart failure", and by the time this develops, changes in the heart are usually regarded as irreversible. This means that the outlook for an affected dog is very poor. Modern drug therapy has lengthened the survival time of affected dogs, but does not cure the condition. The only successful treatment in humans is a heart transplant.

Signs shown by dogs with cardiomyopathy include depression, loss of appetite, breathlessness, coughing, abdominal swelling due to fluid retention (ascites) and cold extremities. In some dogs, unexplained weight loss may be the earliest sign and occasional cases collapse or drop dead. In the wolfhound, almost all cases have a very rapid irregular heartbeat, and this may be appreciated by owners as the heart beats against the chest wall when the dog is lying on its side.

Problems in diagnosis may occur when other diseases cause similar signs eg breathlessness and coughing may be due to pneumonia, weight loss and abdominal swelling may be due to a tumour. Older animals with slowly developing heart disease may have other diseases also and may die of these rather than their heart problem. This is why "cause of death" surveys are of limited value for research purposes.

Accurate diagnosis of heart problems does require specialised veterinary training and expensive equipment, such as electrocardiography (ECG), ultrasound examination (echocardiography) and often chest X - rays.

Not all veterinary practices have suitable equipment for dealing with such large dogs. If there is doubt about whether a dog has heart failure or not, it is essential that further diagnostic tests are carried out because heart failure drugs may have very unpleasant side-effects if given to dogs which do not actually need them.

Drugs are given to relieve congestion and reduce fluid retention (diuretics), to slow the heart if it is beating too fast (digoxin, diltiazem, beta – blockers) and to widen the blood vessels, making it easier for the heart to pump blood through them (vasodilators eg ACE inhibitors.) A new drug called pimobendan has recently been introduced in Europe, which assists the function of the ventricle and also has vasodilator action. This offers promise in the treatment of DCM, but its effects in wolfhounds have not yet been documented.

Causes of cardiomyopathy

The question which everyone wants to answer is "what causes cardiomyopathy?” Unfortunately there are many possible factors involved. Research into DCM was limited until recent years and has tended to follow the same avenues of research as in humans. However, because of the differences between species, it does not necessarily follow that discoveries in one species will apply to others.

Research has concentrated mainly on the following areas:
1) Nutritional problems
Taurine deficiency has been shown to be an important factor in DCM in the cat. Dogs are assumed to manufacture their own taurine, and therefore this is unlikely to be a problem in the dog. Whether this is true of all dog breeds is not known for certain.
L-carnitine deficiency was demonstrated in a family of boxers with DCM in USA and is a rare cause of DCM in children. Absolute deficiency is unlikely but the problem may be with transport or utilisation of carnitine in the body. Treated dogs improved and deteriorated again when supplementation was withdrawn and this caused a great deal of excitement among cardiologists. However, although it is still thought that many dogs with DCM have low levels of L-carnitine in their heart muscle, treatment, even with massive doses, does not usually reverse the disease, as it did in the original boxer family. Carnitine blood and muscle levels are difficult and expensive to measure and the chemical is also very expensive to buy, although it is available in many health food shops.
Other nutritional factors have been said to have protective effects on the heart, but any nutritional approach to heart failure treatment is likely to be unsuccessful as the heart is by then severely damaged.

2) Response to infection
In many cases of DCM, inflammatory cells are found in the heart muscle. This has led to a theory that the disease may be a late stage of an inflammation of the heart muscle (myocarditis). In humans, it has been known for many years that a virus called Coxsackie B is capable of causing heart muscle damage, and more recently another type of virus, adenovirus, has been shown to cause heart damage in children. As far as is known, Coxsackie B does not affect dogs, although there are two canine adenoviruses, causing hepatitis and Kennel Cough. Parvovirus is known to be capable of causing severe heart damage in puppies, but a small study at the Royal Veterinary College did not suggest that this was likely to be a cause in adult dogs. Rarely other types of infectious agent such as Toxoplasma or the organism causing Lymes disease may affect the heart, but there is no evidence that these are of major importance in the dog.
Another possibility is that it is not the virus itself which causes the damage, but the body's immune reaction to it. Anti-heart antibodies have been demonstrated in human cases of DCM but not in normal people. In dogs, it appears that they are present in normal dogs as well as DCM cases. This difference has not so far been explained and the significance of the antibodies is not known.

3) Genetic abnormalities
Ten years ago, less than 10% of total human DCM cases were thought to be familial and these were mainly considered to be related to specific genetic diseases such as muscular dystrophy. More recently, asymptomatic family members of people with DCM have been investigated and now the percentage of genetic DCM cases has risen to 25 - 30%. With the advent of DNA fingerprinting techniques, the actual faulty gene has been identified in a number of human families, but this has complicated the picture because these studies have implicated a number of different genes. The abnormal genes are mainly those that control the formation of the different types of proteins which make up heart muscle.
It has always been interesting to veterinary cardiologists that each breed of dog appears to have its own variant of DCM. This has lent weight to the suspicion that faulty genes may be the most likely cause of the different manifestations of cardiomyopathy in the dog.

Wolfhound DCM
Long-term follow – up studies of heart function in individual dogs of specific breeds are rare in veterinary medicine. The wolfhound study in UK has been conducted over 15 years and has demonstrated that the most unusual feature of the Irish wolfhound is the prevalence in the breed of heart rhythm and conduction abnormalities shown by ECG examination. This suggests that the breed has a problem with the "electrical wiring" which transmits the impulse telling the heart to contract in a co-ordinated manner.
The commonest ECG abnormality is called atrial fibrillation (AF), which affects about 11% of the population. The heart rhythm becomes irregularly irregular and the top chambers, the atria, do not contract. Wolfhounds with heart failure almost always have AF, but initially the heart rate and cardiac ultrasound measurements are usually normal or only slightly increased. We have shown without any doubt that most, if not all, wolfhounds with AF will suffer from gradually deteriorating heart function and will
eventually die of heart failure or sudden death, provided no other disease causes them to succumb in the meantime. This was a surprising finding because death from heart failure is rare in humans with long-standing AF, although heart enlargement does occur and there is an increased risk of stroke. The tendency to develop AF appears to be familial in wolfhounds and many cases have at least one affected parent, therefore breeding from animals with AF cannot be recommended. The average time to death from diagnosis of AF is 2 – 3 years. A few dogs with longer survival times have been identified. It is not known why some individuals survive longer than others, but control of heart rate is important. It is well known that if even a normal heart is made to beat faster than usual for any length of time, this can induce heart failure.

Further research into the mechanisms of the onset of atrial fibrillation may assist us in treatment of wolfhound DCM. It has been assumed previously that once a dog has developed AF, there is no point in trying to restore normal rhythm. However this may not be correct and it is certainly not the attitude of the medical profession, unless the patient is elderly and has other problems. We do know that atrial fibrillation becomes more difficult to reverse the longer it has been present and therefore if any treatment is to be successful, early diagnosis is vital. It is up to wolfhound owners to decide whether or not they are prepared to take steps to achieve this.

The significance of other rhythm and conduction abnormalities demonstrated by ECG is not so clear cut. Premature contractions ("extra beats") and various types of heart block are very common. Some dogs showing these abnormalities will go on to develop atrial fibrillation and DCM, but some will return to normal or continue to have an ECG abnormality, without ever going into cardiac failure. From a genetic point of view, these animals are difficult to classify. However if the dog has a parent or littermate which has developed DCM, or has ultrasound measurements which are outside the normal range for wolfhounds, it is probably safer to assume that they will carry genes for heart disease. Unfortunately nearly all wolfhound families appear to have some individuals affected with heart disease at some stage in their lives and problems may not be observed until dogs have passed breeding age. In the future, DNA testing may offer hope for identifying affected animals, but if research is to be successful, it is vitally important that animals free of disease are also identified. This requires heart examinations to be carried out at least once a year throughout the whole of the dog's life, not just when they are of breeding age.