Degenerative mitral valve disease (DMVD) (previously named myxomatous mitral valve degeneration or mitral valve endocardiosis) is the most commonly encountered cardiopathy in dogs. This disease is characterized by the appearance of nodules on the free edges of the valve and a thickening of the chordae tendinae. As they get bigger, these nodules can fuse and lead to a generalized thickening of the valve. Furthermore, an elongation of the valvular leaflets and a stretching of the chordae tendinae can be observed. The chordae tendinae can rupture, depriving the valve from its support (Figure 1).

This leads to an inadequate coaptation of the leaflets, resulting in a leakage of blood from the left ventricle into the left atrium, called mitral regurgitation (MR). The degree of MR depends on the deformation, the degree of retraction of the leaflets and the status of the chordae tendinae. Even though this disease affects mostly the mitral valve, the tricuspid valve and more rarely the aortic and pulmonic valves can also be affected.

DMVD mostly affects middle-aged small dogs (less than 20 kgs). The Cavalier King Charles Spaniels (CKCS) are particularly predisposed. The prevalence of this disease varies from 14% (non CKCS breeds) to 40% (CKCS). This prevalence increases with age, and can almost reach 100% in CKCS older than 11 years. Large breed dogs, such as the German Shepherd, can also be affected by this disease, albeit less frequently,
CONSEQUENCES

The long term consequences of this MR, depending on its severity, will be dilation of the left-sided cardiac chambers and an increase of pressure in the chamber receiving the regurgitation (the left atrium) (Figure 2).

![Figure 2: Doppler Echocardiography in a dog with DMVD and severe MR. Note the regugitation (arrow) originating from the left ventricle (LV) and filling most of the left atrium (LA). (Photo: Eric de Madron)](image)

This elevation of the left atrial pressure will be transmitted to the pulmonary vessels. When the pressure reaches a certain level, extravasation of serum from the blood vessels into the lung alveoli will occur, a phenomenon called pulmonary oedema (Figure 3).
Cardiac contractility usually remains normal initially, but ends up declining in chronic cases. In 70% of the cases of chronic severe MR, the ever increasing left atrial pressure triggers a vasoconstriction of the small pulmonary arteries, leading to pulmonary hypertension.

**Symptoms**

There are several stages of the disease: stage A (at risk), stage B (MR present, but no symptoms), stage C (MR present, with signs of congestive heart failure), stage D (severe MR with evidence of pulmonary hypertension).

The symptoms associated with onset of pulmonary oedema (left-sided congestive heart failure) include cough, often nocturnal and incessant, rapid and laboured breathing, exercise intolerance, and sometimes fainting. The symptoms associated with pulmonary hypertension include severe exercise intolerance with shortness of breath and sometimes collapse with exercise, and signs of right-sided congestive heart failure such as fluid in the abdomen (ascites), and fluid in the space around the lungs (pleural effusion). In this case, abdominal distension and discordant breathing are noted.

The cardiac auscultation will reveal a characteristic systolic heart murmur over the mitral valve area.

*Figure 3 : Thoracic radiograph from a dog with pulmonary oedema (note the white opacities cranial and caudal to the heart (arrows), due to fluid extravasation in the pulmonary alveoli (Photo : Eric de Madron).*
Prognosis

It is important to note that a great number of dogs with DMVD will remain asymptomatic for a very long time. In a study, the median survival time of 256 dogs with DMVD stage B was 27.6 months. In these 256 dogs, the global mortality rate was 27% over a period of 6.6 years, with a cardiac mortality of 11%. A progression from stage B to C was noted in only 13% of these dogs over that period of time. Another study showed that 82% of dogs in stage B were still asymptomatic 12 months later.

In the dogs with symptoms, the median survival time with treatment was 33 months for the moderate stage C, and 9 months for the severe stage C.

Aggravating factors are:

- the age
- the gender (the disease progresses faster in males)
- the breed (the disease progresses faster in CKCS)
- the degree and rate of progression of the left atrial and left ventricular dilation
- the level of NT-proBNP (a hormone secreted by the cardiac muscle cells)

Treatment

Medical

The treatment aims at helping the heart cope better with the hemodynamic disturbances by fighting the signs of congestive heart failure (diuretics, angiotensin converting enzymes inhibitors (ACEI), spironolactone), the loss of contractility if present (Pimobendan), the pulmonary hypertension if present (Pimobendan, Sildenafil). Some drugs such as Amlodipine can help reduce the amount of regurgitation by reducing the pressure difference between the left ventricle and the left atrium. Arrhythmias, when present, also need to be controlled. There is no specific treatment that can prevent the remodeling of the mitral valve. The use of drugs such as the ACEI in the asymptomatic phase (stage B) may delay onset of symptoms, but the efficacy varies among breeds (ineffective in CKCS).

Surgical

In human medicine, treatment of mitral regurgitation is essentially surgical, with mitral valve repair or replacement, requiring open heart surgery and by-pass. Such approaches are not readily available in veterinary medicine. Only a few centers (mostly in Japan) are doing it in dogs. Therefore, medical management remain the only option at this stage. However, this is likely to change in the near future.

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References:


Dog photo: www.animalplanet.com