INFO: Hypertrophic Cardiomyopathy (HCM)

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WHAT IS HCM?

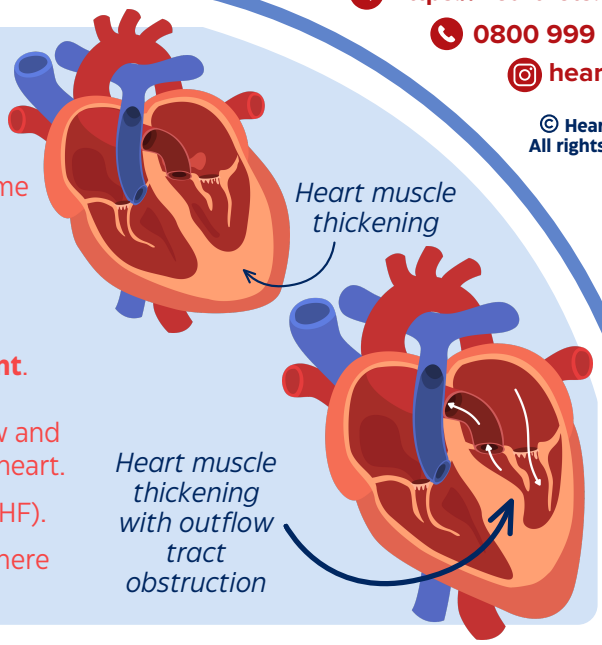
A disease affecting the heart muscle, causing it to become **thicker and stiffer** over time.
The thickening (hypertrophy) can be **focal** (small areas) or **global** (throughout).

The reduced ability of the heart muscle to **relax** to accept blood leads to **chamber enlargement**.

Enlargement of the left atrium leads to reduced blood flow and increased risk of **blood clots** (thrombi) forming within the heart.

Eventually, patients develop **congestive heart failure** (CHF).

Some patients have **outflow tract obstruction** (OTO), where there is a narrowing as blood leaves the heart.



HOW DO YOU DIAGNOSE HCM?

- Some patients have **heart murmurs** ('whooshing' noise heard with a stethoscope, but many with HCM do not have a murmur. **The presence of a murmur is not a diagnosis.**
- **ProBNP blood test:** screening for heart stretch – abnormal results indicate that a heart scan is required.
- **Echocardiography** (heart ultrasound scan) is used to look at the heart structures and function, diagnose HCM and look for OTO.
- **ECG:** monitors electrical activity, checking heart rate and rhythm and changes in waveform.
- **Xray** can be used to look for fluid in the lungs or lung disease.

WHAT CAN HAPPEN AS HCM PROGRESSES?

- **Myocardial infarction** (MI): thickening of the heart muscle restricts blood supply to the inner layers. Loss of blood supply (and oxygen) causes these parts to 'die off'. This can be seen as areas of thinning and reduced motion.
- **Arrhythmias** (abnormal rhythms) can develop as the heart muscle becomes more diseased or scarred. Dangerous rhythms can increase the risk of sudden death.
- **Aortic thromboembolism** (ATE) occurs if a blood clot (thrombus) formed in the heart gets stuck at the narrowing of the aorta in the abdomen, where it divides to supply the hind legs. This prevents blood supply getting to the legs.
- Chronic blood supply issues, MI and scar tissue formation leads to **thinning** of the (previously thickened) heart muscle and **severe reduction in pump function** in end stages of HCM.
- CHF is commonly seen as **pulmonary oedema** (build up of fluid in the lungs), but can also cause **pleural effusion** (build up of free fluid in the chest cavity, around the lungs).

WHAT ARE THE CLINICAL SIGNS?

Speak to your vet if you think your pet may have any of these signs.

Patients can have **no clinical signs** and still have underlying structural changes of HCM. Cats are particularly good at hiding any sign of problems until disease is advanced.

Clinical signs can include:

- Lethargy, withdrawl behaviour
- Reduced exercise tolerance / play
- Increased respiratory rate or effort / open-mouthed breathing (particularly during exercise or exertion). If sustained: **this is a genuine emergency**
- Cough
- Weakness or collapse
- Decreased appetite
- Sudden loss of / change in hind limb function or lameness – **this is a genuine emergency**

HOW IS HCM TREATED?

Treatment in HCM is aimed at two main aspects of HCM:

Blood clot prevention: although we cannot prevent clots forming entirely, we try to reduce the likelihood of them forming (clopidogrel, rivaroxaban or aspirin).

This is started when there is evidence of enlargement of the left atrium and reduced speed of blood flow, or if we can see clots already forming within the heart.

CHF: diuretics (frusemide or torasemide) remove pulmonary oedema or pleural effusion.

These are started when there is evidence of CHF on echocardiography / xrays and clinical signs (increased sleeping respiratory rate (SRR).

In some specific cases we may add an ACE inhibitor (improves circulation), anti-arrhythmic/rate control drug (treats arrhythmias) or pimobendan (improves pump function).

See our website for information on heart testing schemes

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