Case Study 2

Smokey

Management of hyperthyroid cats with concurrent disease

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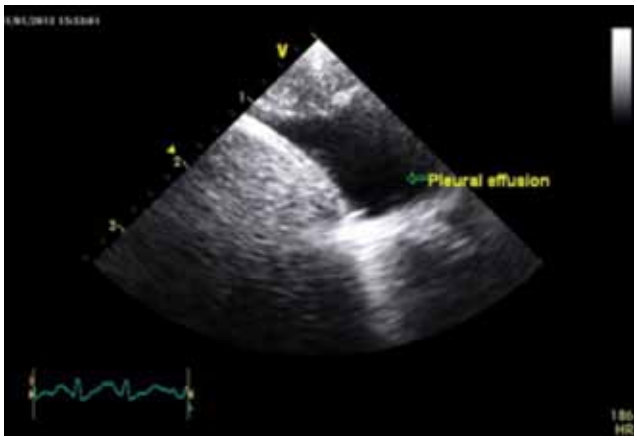
Smokey was a 10 year old female neutered domestic short hair who presented for further investigation of recurrent pleural effusions and associated dyspnoea. These were suspected to be secondary to congestive cardiac failure, which had been diagnosed some months previously and was managed medically with diuretics, an angiotensin converting enzyme inhibitor and aspirin. Despite these combined therapies, pleural effusions were still a frequent occurrence and compromising Smokey's quality of life.

On presentation, Smokey had mild-moderate tachypnoea, with a shallow rapid respiratory pattern. Pulmonary auscultation identified muffled lung sounds ventrally bilaterally, giving a high suspicion for a pleural effusion. There was a moderate tachycardia, with a grade III/VI parasternal systolic heart murmur and a gallop sound. Other salient clinical examination findings included a large (12-15 mm diameter) mobile goitre, palpable dropping into the thoracic inlet.



Moderate volume pleural effusion.

Thoracic ultrasonography confirmed the presence of a moderate volume pleural effusion; 95 mls of a grossly milky fluid was drained by thoracocentesis; analysis was consistent with a chronic chylothorax.

Echocardiography was consistent with moderate to severe hypertrophic cardiomyopathy (HCM) with severe diastolic dysfunction and marked bilateral atrial enlargement. Hypertrophic cardiomyopathy may be either primary or secondary (to e.g. hyperthyroidism, hypertension, acromegaly). The severity of Smokey's echocardiographic findings made the presence of primary hypertrophic cardiomyopathy likely, with or without the presence of exacerbating secondary factors. Total T4 (thyroxine) was found to be elevated (76.6 nmol/l; ref. 15-60 nmol/l); confirming the presence of hyperthyroidism.

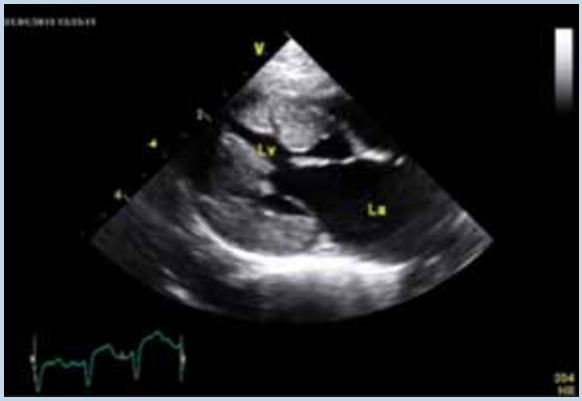
Gross appearance of a chylous effusion; this may vary from milky white to blood tinged.

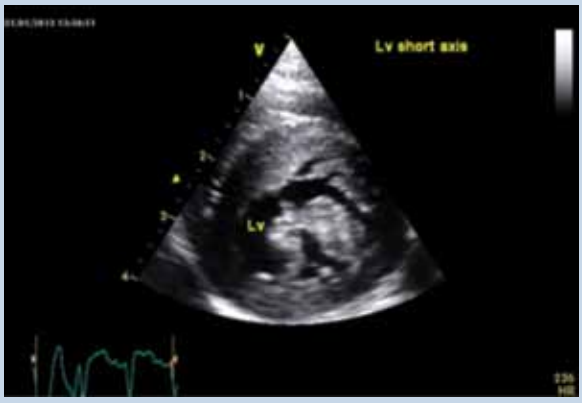
Prior to devising a management regime, haematology and biochemistry were performed and identified a moderate increase in alanine aminotransferase activity and marginal increase in alkaline phosphatase activity (134 iu/l; ref. 15-45 iu/l and 62 iu/l; ref. 15-60 iu/l respectively), as is found in >90% of hyperthyroid cats. There was also a mild increase in urea, with low creatinine (13.5 mmol/l; ref. 6.5-10.5 mmol/l and 99 µmol/l; ref. 133-175 µmol/l respectively); likely consistent with a pre-renal azotaemia, although urine concentrating ability could not be accurately assessed due to prior diuretic administration. There was a mild hyperphosphataemia (1.78 mmol/l; ref. 0.95-1.55 mmol/l), which is not an uncommon finding in hyperthyroidism (found in approximately one third of cases and thought to be secondary to altered bone metabolism in the hyperthyroid state), although this finding may also increase suspicion of masked chronic kidney disease.

Problems identified in Smokey included:

- Recurrent chylothorax
- Hypertrophic cardiomyopathy
- Uncontrolled hyperthyroidism

The pivotal feature to control in this case was the hyperthyroidism. Without adequate control of the hyperthyroid state, we could not expect to achieve good control of the congestive heart failure and subsequently the recurrent pleural effusions.





Echocardiographic images demonstrating severe HCM.

Felimazole® 2.5 mg PO BID was prescribed for management of the hyperthyroidism. Further adjustments to the therapeutic protocol for congestive cardiac failure were also made (increased diuretic dose, further anti-platelet aggregation therapies, addition of an inodilator), to reduce the risk of repeat manifestation of congestive failure during the period of stabilisation of the hyperthyroidism.

Over the first ten days of therapy, Smokey exhibited a significant improvement in demeanour. Clinical examination identified a normal respiratory pattern with resolution of both the tachycardia and gallop sounds. There was no ultrasonographic evidence of a pleural effusion.

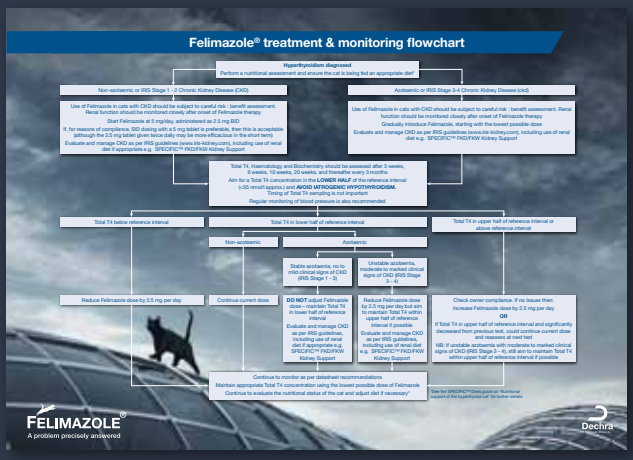
Reassessment following three weeks on treatment demonstrated on-going clinical improvement and a pleasing weight gain, consistent with good control of the hyperthyroidism. Repeat haematology and biochemistry at this point demonstrated the presence of a moderate azotaemia (urea 25.3 mmol/l, creatinine 357 µmol/l). Serum total T4 assessment demonstrated biochemical hypothyroidism (<12.9 nmol/l). Concurrent diuretic therapy precluded the assessment of urine specific gravity to exclude or confirm a pre-renal azotaemia. However, the azotaemia was considered likely multifactorial in origin; both reflecting unmasking of renal disease with control of the hyperthyroidism (renal) and also subclinical dehydration with more aggressive diuretic therapy (pre-renal).

This development added chronic kidney disease to Smokey's problem list. In the management of hyperthyroidism, mild biochemical hypothyroidism (total T4 10-15 nmol/l) is acceptable in the absence of azotaemia or clinical signs of hypothyroidism. However, in both of these circumstances over-suppression of the thyroid gland is not appropriate, therefore a reduction in the Felimazole dose was advised.

A Felimazole dose of alternating daily doses of 2.5 mg SID and 2.5 mg BID suited Smokey purrrfectly! She was clinically well and euthyroid (total T4 18.4 nmol/l). A moderate azotaemia persisted, although was biochemically less marked (urea 20.5 mmol/l, creatinine 207 µmol/l). This is exactly the sort of situation in which we would now make use of the newly licensed 1.25 mg Felimazole tablets. Although, historically, alternating daily doses have enabled achievement of clinical and biochemical euthyroidism,

the opportunity to administer a consistent daily dose (i.e. in this case 2.5 mg AM, 1.25 mg PM) should enable more uniform thyroid suppression and thus better control of the clinical disease. In the presence of stable azotaemia, management of hyperthyroidism should be aimed at achieving a total T4 value in the lower half of the reference interval, with concurrent appropriate therapy for chronic kidney disease. Although poor control of hyperthyroidism may result in apparent reduction in severity of the azotaemia, this may also contribute to progression of the chronic kidney disease with time, due to the damaging effects of increased glomerular filtration rate associated with the hyperthyroid state.

Following establishment of stable euthyroidism and congestive cardiac failure, we were able to appropriately stage Smokey's chronic kidney disease. Plasma creatinine was serially assessed and was consistently IRIS stage II (creatinine 140-249 µmol/l). IRIS guidelines were used to further substage (based upon hypertension and proteinuria) and manage the chronic kidney disease. As expected with primary hypertrophic cardiomyopathy there was echocardiographic progression over the following months, although with the tight control of hyperthyroidism afforded by flexible dosing with Felimazole, and adequate medical therapy of the congestive cardiac failure, we were able to eliminate the recurrence of pleural effusions for a respectable duration of time; allowing Smokey to go about her normal day-to-day cat behaviour without the requirement for repeat thoracocentesis.



For further advice on medical management of hyperthyroidism with Felimazole please refer to the 'Felimazole treatment & monitoring flowchart' produced by Dechra Veterinary Products. This includes information on the management of hyperthyroidism cats with concurrent chronic kidney disease.

The treatments and doses described in this case study are entirely at the discretion of the author and are based on their own considerable clinical experience. It is the responsibility of individual prescribing veterinary surgeons to ensure that they comply with local veterinary medicine regulations.

Felimazole contains thiamazole UK POM-V IE POM
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