PURINA® PRO PLAN® VETERINARY DIETS

This case report demonstrates the benefits of PURINA® PRO PLAN® VETERINARY DIETS Canine HP Hepatic in the nutritional management of hepatic disease in the dog

Canine HP Hepatic in the nutritional management of hepatic disease

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Introduction

Photo 1: Teddy

Teddy *(photo 1)*, a 12-year-old male neutered Labrador, was referred to us for investigation and management of a **low-grade jaundice**. Teddy, the only animal in the household, lived in a house with access to a garden. He had been given a dry food from a specialist pet shop. Acquired as a puppy, Teddy was vaccinated every year and wormed regularly. He wore an anti-flea/tick collar which was changed every six months.

Clinical examination

Teddy was in good general condition, although slightly overweight (body condition score 5/9). His coat was satisfactory. His temperature was normal (38.9°C) and had an estimated dehydration of <5 %.

On cardiocirculatory and respiratory examination, **the mucosae were pale and slightly jaundiced**. Relaxed abdominal palpation found **discomfort in the cranial abdomen**, with uniform **hepatomegaly and mild induration.**

Further investigations

Urinalysis after collection by cystocentesis indicated severe tubulopathy in the absence of associated hyperglycaemia (good urine concentration (urine specific gravity 1.040, pH 7), bilirubin 1+, protein 3+, glucose 4+, urobilinogen negative, epithelial cells 2+, casts 2+, protein debris 2+). Electrolytes were normal and plasma biochemistry detected a **jaundiced plasma**, cytolysis, **significant cholestasis (ALT, ALKP) and inflammatory disease (elevated protein).**

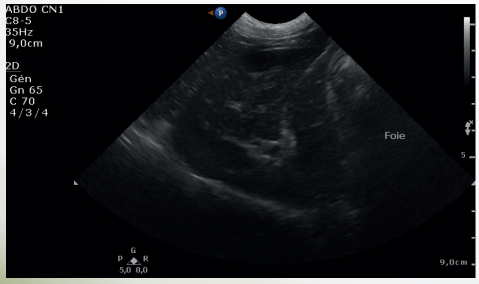

Photo 2

A complete blood count confirmed no abnormalities of the different cell lines. Abdominal ultrasound *(photo 2)* confirmed the absence of ascites and suggested **severe diffuse hepatopathy** (with hypoechogenicity suggesting an acute condition), and a **perihepatic peritonitis**. Other associated findings were **chronic nephropathy (irregular kidneys)** and **gastritis**. Leptospirosis detection by PCR in blood and urine was negative.

Principal diagnostic hypotheses

The epidemio-clinical context and further investigations suggested:

1. **Hepatopathy, fairly acute in the current context,** without ruling out an underlying chronic inflammatory process
2. **Chronic nephropathy with tubulopathy** (creatinine upper limit, glycosuria, irregular kidneys)

As both organs were affected simultaneously, it was not possible to rule out drug intolerance or chronic inflammatory disease of bacterial origin (leptospirosis, etc.) or related to immune dysfunction (glomerulonephritis/chronic hepatitis). A neoplastic process was unlikely but could not be ruled out at this stage.

Management and definitive diagnosis

The initial management comprised fluid therapy (NaCl 0.9% supplemented with potassium 100 ml/kg/d (3 ml/kg/h)), a proton pump inhibitor (omeprazole 1 mg/kg/d PO), antibiotic therapy (amoxicillin and clavulanic acid 20 mg/kg/d SC), and an antiemetic (metoclopramide 0.5 mg/kg/8h SC).

After 24 hours of in-patient care, Teddy's clinical condition deteriorated. Despite treatment, he had become anorexic and was vomiting. He was also overhydrated. Oliguric renal insufficiency (leptospirosis in origin) was suspected. Creatinine levels were elevated (157.5 µmol/l). Fluid therapy was reduced to 1 ml/kg/h.

Anorexia and marked depression were still present on the following day. Pulmonary radiography confirmed the absence of pulmonary oedema. A naso-oesophageal feeding tube was inserted to assist gradual feeding. Enteral nutrition led to a clinical improvement and normal hydration. An improvement in renal function was observed, but the **hepatic abnormalities (elevated ALT, ALKP, plasma still jaundiced) persisted.**

Three injections of vitamin K1 were given (2 mg/kg IV). Three liver biopsies and three biopsies of the left kidney were taken under general anaesthetic.

After four days of inpatient care, Teddy showed some clinical improvement. Follow-up biochemistry showed a further increase in ALKP, while the other parameters (ALT, total bilirubin and creatinine) remained stable compared with the previous results.

The follow-up ultrasound was unchanged. Feeding by naso-oesophageal tube was continued until Teddy was eating independently again. On the evening before discharge, Teddy spontaneously resumed eating.

Teddy was sent home with the following prescriptions: Zentonil Advanced (S-adenosyl methionine and silybinin) 800 mg/d (24 mg/kg/d) for 1 month, Ursolvan (ursodeoxycholic acid) 500 mg/d (15 mg/kg/d) for 3 months, omeprazole 40 mg/d (1.2 mg/kg/d) for 1 month, Kesium 500 mg/12h (amoxicillin and clavulanic acid, 15 mg/kg/12h) for 10 days, Dermipred (prednisolone) 10 mg/d (0.3 mg/kg/d) for 5 days and then once every 2 days for 10 days.

Histopathology revealed chronic multifocal hepatitis of marked severity (lymphoplasmocytic and neutrophilic infiltration) with large numbers of pigmented macrophages and moderately severe accumulations of copper at multiple sites in the liver. Quantitative determination of copper in biopsies suggested copper accumulation secondary to chronic hepatopathy. Leptospirosis serology was negative. The definitive diagnosis was therefore chronic multifocal hepatitis of marked severity with moderately severe secondary accumulation of copper.

On receiving the results, a further examination was performed. Teddy's general condition was good. The biochemistry profile was extended and treatment was revised. Teddy's appetite at this time was good, with homemade food divided into three meals initially. No digestive upsets were reported, despite nausea and impaired appetite (25 to 50% reduction in spontaneous intake of his regular diet). Urinalysis after collection by cystocentesis showed a urine SG of 1.035, pH 6, protein 1+, and no glucose, bilirubin or urobilinogen in the urine. A SNAP cPL test was performed and was negative, confirming the absence of acute pancreatitis. The fructosamine level of 302 (<360) confirmed the absence of diabetes. The plasma biochemistry profile found elevated levels of ALT (895 U/l), ALKP (825 U/l) and bilirubin (12.8 µmol/l). Creatinine had dropped (106.2 µmol/l).

The current treatment was continued, except for the antibiotics. Vitamin E was added at 500 mg per day (i.e. 15 mg/kg/d), given in the morning after fasting. With a four-day transition, Teddy's diet was changed to **Canine HP Hepatic** (350 g per day).

After a few days on his new diet, Teddy's general condition was good. He was fit, healthy and playful. Plasma biochemistry showed a marked improvement in cytolysis with a reduction in ALT (390 U/l), an increase in cholestasis (elevated ALKP (1,623 U/l)), disappearance of any visible jaundice, highly lipaemic plasma even after 12 hours of fasting and a complete return of normal renal function (creatinine 55.7 µmol/l).

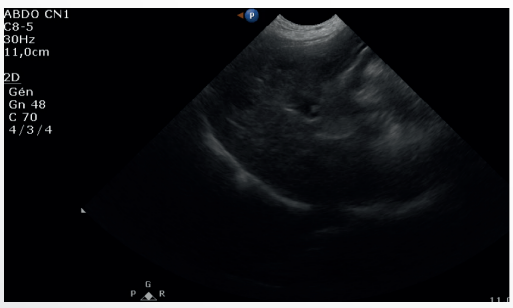

Photo 3

Follow-up

After one month, Teddy was doing very well.

His owners reported no digestive upsets. His food intake was adjusted. His weight was 31.5 kg, body condition score 6/9 and muscle mass score 1/4. His coat was satisfactory. A general clinical examination identified no notable abnormalities. Abdominal ultrasound *(photo 3)* showed an improvement in the intestinal tract images compared with the last check-up, with a uniform, hyperechoic liver. Urinalysis after collection by cystocentesis showed a urine SG of 1.035, pH 6, protein 1+, and no urobilin or bilirubin. The (fasting) biochemistry profile showed a reduction in ALT (103 U/l), ALKP (1061 U/l) and GGT (19 U/l), plus a raised cholesterol (10.9 mmol/l). Feeding with **Canine HP Hepatic** (340 g per day) and concurrent treatment was continued, with the exception of Zentonil and Kesium.

After two months, Teddy's overall health was good.

Urinalysis showed a urine specific gravity of 1.018 and pH of 9, with no other abnormalities. The complete blood count and blood smear were normal. Plasma biochemistry three hours after the last meal showed raised cholesterol (15.7 mmol/l) and a reducing ALKP (636 U/l). On abdominal ultrasound *(photo 4)*, the liver showed poor uniformity with 8 mm of hyperechoic biliary sludge associated with a slight mineralisation (presence of shadow cone).

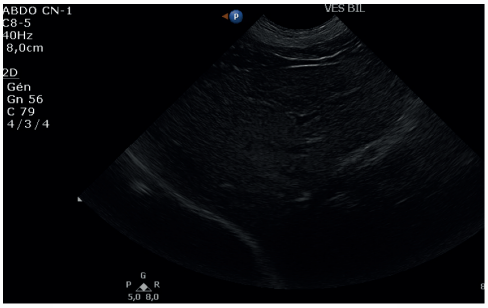

Photo 4

Discussion and conclusion

Throughout the study, Teddy showed no reluctance to eat the **Canine HP Hepatic dry food**. He ate well, without any adverse effects. The change of diet caused no problems. The owners reported that the diet was effective, and their dog's general health was excellent. Indeed, they were keen to adopt the diet permanently if it was made available on the market. The owners would recommend the diet on veterinary advice for an animal with hepatic insufficiency.

The diet seemed effective, and as a strong point, it was very palatable. It was also well-tolerated. I believe this is a good diet to use in animals with hepatic insufficiency or copper-associated hepatitis in particular.

Nestlé PURINA would like to thank Dr Charru for providing the details of this case.