



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



Belle, a seven-year-old female neutered Great Dane, was brought to the clinic with a four to five day history of anorexia. Belle lived in a house with a garden, and was the only animal in the household. She had been eating a dry food from a specialist shop (Dog Chow adult, 730 g per day). She had been fully vaccinated, wormed and treated for external parasites.

Medical history:

- Previous year: Removal of a 6.1 kg splenic mass. Histology suggested a splenic haematoma (based on cells represented in sample, no tumour cells)
- History of uterine cystic glandular hyperplasia and pseudopregnancy with lactation, treated medically (most recently a few months previously)
- Recently: biopsy of a lipoma on the right hock

Clinical examination

- Belle's had a weight of 65 kg, a body condition score 4/9, a muscle mass score 3/4, a dehydration estimated at 5% and a mild hyperthermia (39.4°C)
- Cardio-circulatory and respiratory examination: mucosae pale pink, capillary refill time < 2s, tachycardia 170 bpm, femoral pulse clear and synchronous, no other abnormalities on heart and lung auscultation was normal. Pain was detectable in cranial abdomen on abdominal palpation, with a cranial mass but no enlargement of peripheral lymph nodes were detected

Belle's general condition was not good.

Futher investigations

- <u>Urinalysis by cystocentesis:</u> slightly low urine specific gravity (1.025), pH 7, protein 1+, rest of test strip negative
- Plasma biochemistry: creatinine 66.4 μmol/l, ALKP 333 U/l, ALT 27 U/l, total bilirubin 5.8 μmol/l, total protein 76 g/l, albumin 27.4 g/l, cholesterol 4.6 mmol/l, triglycerides 0.55 mmol/l, GGT 8 U/l, glucose 7.3 mmol/l, urea 4.8 mmol/l, post-prandial bile acids 3.6 μmol/l

- Normal electrolytes: Na 151 mmol/l, K 4.4 mmol/l, Cl 118 mmol/l
- Fructosamine 197 μmol/l (<360)
- <u>Complete blood count</u>: mild neutrophilia of 17.42 x 109/l WBC, incl. 14.24 neutrophils (3.0–13.6), 0.54 monocytes and 2.64 lymphocytes, mild anaemia with haematocrit 28% (35–55), no other erythroid lineage abnormalities (6.51 x 1012/l RBC, Hb 122 g/l, MCH lower limit 18.7 pg (19.5–24.5), MCHC 30.5 g/dl), platelets normal 307 x 109/l
- <u>Blood smear</u>: neutrophilic macrocytic leukocytosis with reactive neutrophils, left shift, reactive monocytes, suspected thrombocytosis, no erythroid lineage abnormalities
- <u>Abdominal ultrasound</u>: low abdominal fat, no ascites
 - Liver: large non-uniform hollow hepatic mass touching all hepatic lobes and including the gall bladder
 - Gastrointestinal tract: stomach wall 1.4 cm with thickening of mucous layer, wall of duodenum 4.03 mm, jejunum 2.4 mm, ileum 3.8 mm, layered structure intact
- Adrenal glands: thickness 4.91 mm (L) and 6.43 mm (R) (seen 4–6.5 mm), shape and layers intact
- Kidneys: length 7.5 cm (L) and 7.6 cm (R), clear corticomedullary distinction. Bladder wall slightly thickened (6.7 mm) but bladder within normal limits, empty
- Spleen, pancreas, bladder normal

Biochemistry follow-up

PARAMETERS	30/12	31/12	Normal values
Post-prandial bile acids (µmol/l)		3.6	< 50
Albumin (g/l)	27.4		26 - 40
ALT (U/I)		27	< 70
Bilirubin (µmol/l)		5.81	< 10.3
Cholesterol (mmol/l)	4.6		1.15 - 6.57
Creatinine (µmol/l)	66.4		42.5 - 133
Glucose (mmol/l)	7.32		3.3 - 6.7
GGT (U/I)	8		< 10
ALKP (U/I)	333	337	20 - 270
Total protein (g/l)		76	55 - 70
Triglycerides (mmol/l)	0.57		< 0.58
Urea (mmol/l)	4.83	-	1.67 - 10.0
Sodium (mmol/l)	151		
Potassium (mmol/l)	4.4		
Chloride (mmol/l)	118		

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Conclusions

Presence of large hepatic mass with no current signs of abdominal metastasis:

- Normal clotting times: QT 14 s (7-15) and APTT 94 s (54-94)
- <u>Hepatic cytology</u> inconclusive
- Liver biopsies under laparoscopy (VetHisto): awaiting results
 - Liver surface irregular, hepatomegaly with small area of normal appearance
 - Stomach and falciform ligament pushed to the left
 - Slightly ulcerative area on left side of diaphragm => Biopsy of this area
- Bacteriology and test for mycobacteria (Pasteur): negative (results received 14/01)

Abdominal ultrasound images



Hepatic mass



Stomach



Management and definitive diagnosis

Initial management:

- Fluid therapy: NaCl 0.9% supplemented with potassium 120 ml/kg/d (3 ml/kg/h)
- Antibiotic therapy: enrofloxacin 5 mg/kg/d IV, amoxicillin and clavulanic acid 20 mg/kg/d SC, metronidazole 15 mg/kg/12h PO
- Analgesia: morphine 0.2 mg/kg/4h IV
- Antiemetic: metoclopramide 0.5 mg/kg/8h SC
- Bile thinner: ursodeoxycholic acid 10 mg/kg/d PO

Discharge after three days of in-patient care, awaiting results:

- Haematocrit 25%
- Antibiotic therapy: amoxicillin and clavulanic acid 12.5 mg/kg/12h PO for three weeks, metronidazole 10 mg/kg/12h PO for three weeks => Antibiotics discontinued after 12 days' treatment on receipt of results
- Antiemetic: metoclopramide 0.5 mg/kg/8h PO for four days
- Fed with Canine HP Hepatic, 600 g per day, with a dietary transition over four days

Treatment was revised after two weeks on receipt of histology results Belle's condition was satisfactory, she ate nearly 90% of her ration of Canine HP Hepatic.

Histopathology (see results):

- Biopsies of liver demonstrated infiltration by an undifferentiated tumour
- Biopsy of hyperplastic area of diaphragm showed papillary mesothelial hyperplasia of marked severity
- Non-steroidal anti-inflammatory: Cimalgex (cimicoxib) 2 mg/kg/d with meals. Antiemetic: metoclopramide 0.5 mg/kg/8h SC resumed for impaired appetite and nausea

Despite treatment, Belle became anorexic and appeared depressed. Treatment was discontinued. Phytotherapy was initiated (Desmopar and L-glutamine) but without success.

Belle died of abdominal haemorrhage one month after the onset of symptoms.

Discussion and conclusion

Throughout the study, Belle showed a lack of appetite, obviously related to her progressive, invasive abdominal disease. Even so, she continued to eat Canine HP Hepatic willingly and with a good appetite.

In this case it is difficult to gauge precisely the effect that the diet had on the progression of the disease. Belle unfortunately failed to complete the study due to a severe progressive hepatic disease (an aggressive hepatic tumour of unknown origin). However, despite her condition and her invasive neoplastic hepatic mass, Belle has accepted and ate well the diet, reflecting its high palatability.