



HEPATIC LIPIDOSIS WITH COMPLICATIONS

Case Report
Lauren Matzuka, DVM

REVIEWED BY
DR. TODD DEPPE, DVM, DIPLOMATE ACVIM

Signalment:

“Hugo” 12 year old MN DSH with BCS 3.5/9

History:

Hugo has never had a good appetite since he was obtained 2 months ago from a shelter. He had lost 3 pounds during this period. Over the past 5 days he has had to be force fed A/D mixed with water. Hugo has had intermittent vomiting occurring 30 minutes after being fed. His primary veterinarian noted that he was jaundice 2 days prior to presentation to VMSG. Blood work revealed an anemia (Hct 26%, Tbil 9.5, ALP 182, CPK 1028). His urine was orange, turbid, containing protein and bilirubin, however, it was concentrated (USG 1.053). Clavamox and metoclopramide were initiated with no resolution of symptoms. Hugo was thus referred to VMSG for abdominal ultrasound and feeding tube placement.



Clinical exam:

On presentation, he had mild generalized jaundice (pinna, conjunctiva, sclera,), a grade II/VI systolic parasternal heart murmur, hepatomegaly, and was 5% dehydrated.

Blood work:

In-house blood work revealed PCV 25%, TS 6.7 g/dl, severely icteric serum, and a respiratory alkalosis (pH 7.466, pCO₂ 24.8).

Diagnostic imaging:

Thoracic radiographs were unremarkable. Abdominal ultrasound revealed diffuse hyperechoic hepatomegaly (figure 1). An ultrasound-guided fine needle aspirate of the liver revealed cytoplasmic vacuoles within more than 80% of the hepatocytes with no evidence of neoplasia or inflammatory infiltration. (figure 2).

Figure 1): Ultrasound of the liver revealed a hyperechoic, enlarged liver

Figure 2): Picture on the left is normal liver. The picture on the right is a fatty liver.

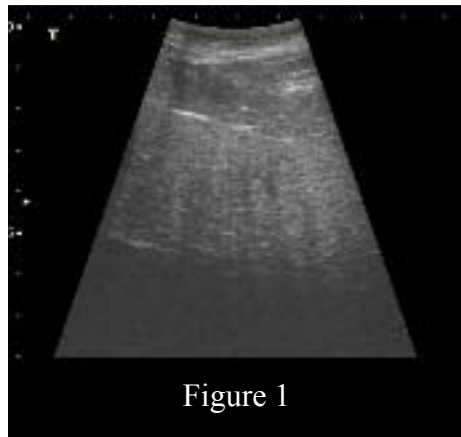


Figure 1

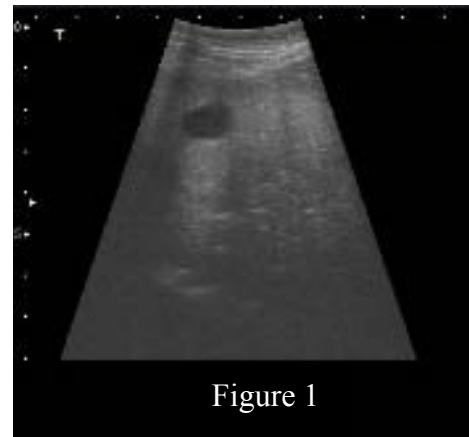


Figure 1

Diagnosis:

Hugo’s clinical symptoms, anemia, hyperechoic hepatomegaly, and fine needle aspirate findings are all consistent with hepatic lipidosis.

Treatment/Management:

The dehydration was addressed with Normosol R qs 20 mEq KCl + vitamin B complex. An esophagotomy tube was placed under sedation with a lateral thoracic radiograph confirming

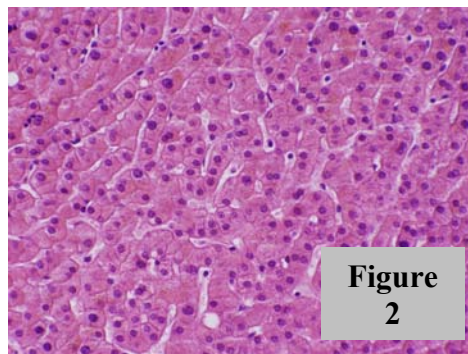
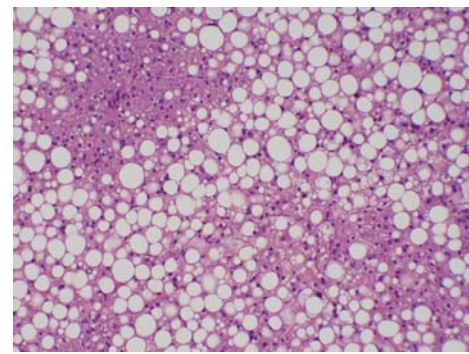


Figure 2



accurate placement. By e-tube Hugo was started on a slurry of A/D at 25% of his caloric intake dispersed into four feedings a day. If Hugo did not vomit, his caloric intake would be increased 25% each day until he reached 100%. Famotidine BID and metoclopramide TID were crushed and given by e-tube: Denosyl (SAME) was started for its antioxidant, anti-inflammatory properties, and helps restore hepatocyte dysfunction. Because of Denosyl’s enteric coating, it is best absorbed orally, however can be given by e-tube if the dose is increased by 50%. Ursodiol capsules were given whole by e-tube, to prevent biliary stasis secondary to hepatocyte swelling. Both Denosyl and ursodiol were discontinued because of recurrent vomiting.

Complications:

Just prior to Hugo going home he developed a fever of 103.8 and mucopurulent discharge around the e-tube site. In-house cytology of the discharge revealed degenerative neutrophils with gram-negative rods. Clavamox and Baytril were given by e-tube. His e-tube site was cleaned and rewrapped twice a day until the discharge had resolved.

After starting the antibiotics Hugo began to vomit after his e-tube feedings. Differentials for his consistent vomiting were decreased GI motility, improper e-tube feedings (too much, too fast, not flushing between medications/feedings), side effects of the antibiotics, pancreatitis, hyperthyroidism, or inflammatory bowel disease. The antibiotics were given after the food, and his feedings were decreased in quantity but increased in frequency to six times a day to maintain caloric intake at 25%. Hugo continued to vomit, so Anzemet injections were initiated.

One week later Hugo was still vomiting, and his blood work revealed progression of his liver disease: His total bilirubin had increased from 9.5 to 16.6, white blood cells were 17,200, and he had a regenerative anemia of 16% (originally 25%). Hepatic lipidosis can cause anemia secondary to hypophosphatemia (normal in Hugo's case) or possibly by depletion of glutathione (GSH). Progression of anemia in Hugo's case was most likely from severity of hepatic lipidosis with a possible secondary coagulopathy. A blood transfusion was recommended, but for financial concerns the owner decided to monitor the anemia. The following day his PCV dropped to 15% and TS 8.0 g/dl. Two days later Hugo's PCV was 14%, TS 6.6, and severely icteric serum. He was blood typed as blood group A, cross-matched, given 2 mg/kg SQ diphenhydramine, and received 25 mls of feline packed red blood cells with no complications. Post-transfusion PCV and TS was 20% and 7.7 respectively. Vitamin B12 and B complex injections were given SQ.

Hepatic lipidosis cats usually do not continue to vomit 1 week post e-tube placement. A secondary disease, such as inflammatory bowel disease (IBD) was suspected, and the owner chose to treat medically rather than diagnose this disease definitively with upper GI endoscopy and biopsy of the intestine. Hugo was switched to Z/D gruel, given dexamethasone 0.3 mg/kg SID SQ for 10 days, and vitamin K 1.5 mg/kg SQ one time. He was continued on the Clavamox, Baytril, famotidine, metoclopramide all by e-tube, and Anzemet SQ. Hugo stopped vomiting, and his feedings were increased by 5 mls daily until 100% caloric intake was attained.

Four days later his PCV was maintaining at 18.5% and TS 5.5 g/dl with less icteric serum. SAME 200 mg PO SID and ursodiol 30 mg by e-tube were started since he had no vomiting. The Clavamox and Anzemet were discontinued. Baytril was continued for three weeks in case Hemobartonella (*Mycoplasma* sp.) was causing the anemia. He was switched to prednisolone 5 mg PO BID, continued on the famotidine, metoclopramide, and Z/D by e-tube. He developed an upper respiratory infection, and was started on BNP OU QID for 2 weeks.

Follow-up:

By week four Hugo was eating on his own, and his blood work had improved (regenerative anemia 18%, normal WBC, Tbil 0.9, ALT 336). The metoclopramide was discontinued, and he was started on weekly vitamin B12 1000 mcg SQ. Famotidine was continued for 1 more week; and the prednisolone would be weaned in 1 month to 5 mg PO EOD. The e-tube feedings were stopped for 1 week, during which time he ate well on his own. Five weeks after being diagnosed with hepatic lipidosis his e-tube was removed, and his weight was the same as when he first presented.

Prognosis:

The prognosis for hepatic lipidosis is good if treated aggressively, with 80% or more surviving. Almost all cats recover if they survive the first 2-3 days.

Pathophysiology:

Hepatic lipidosis is a metabolic disorder where triglycerides accumulate in the hepatocytes. In a normal liver only 5% of the hepatocytes have triglycerides, whereas with hepatic lipidosis 80% or more have triglycerides. Obese cats are affected by not eating for several days or eating ½ their normal diet over several weeks causing them to mobilize free fatty acids from their adipose tissue by lipolysis. Hormone-sensitive lipase promotes lipolysis, and is increased by norepinephrine, epinephrine, growth hormone, corticosteroids, thyroxin, and glucagons. Free fatty acids are transferred to the liver where they can become triglycerides, phospholipids, lipoproteins, cholesterol, or undergo beta-oxidation to make ATP. Triglycerides cannot leave hepatocytes if production is more than export. It has been hypothesized that hepatic triglyceride accumulation can occur if there is decreased beta-oxidation, or not enough apoproteins available to transport triglycerides out of the hepatocytes.

Discussion:

When rehydrating a hepatic lipidosis cat, lactate and dextrose containing fluids should be avoided, and vitamin B should be added to the fluids. Hepatic lipidosis cats have been associated with high lactate levels, and dextrose can potentiate hepatic triglyceride accumulation. These cats are not eating thus are not absorbing vitamin B or vitamin K. In less than 7 days cats can become vitamin K deficient. Vitamin K is needed in making the clotting factors II, VII, IX and X, thus these cats can develop coagulopathies. Esophagotomy tubes are recommended, and should be maintained until the patient is able to take in their full daily caloric intake (average 4-8 weeks). Esophagotomy tubes are more long-term than nasogastric tubes, which can only stay in for 3-7 days, and are safer than gastrostomy tubes. Everything should be given through the e-tube and small amounts of food should be given over the first few days (starting with 25% daily caloric intake over 3-4 feedings and increasing slowly). If the cat is not improving within 2-4 weeks or continues to have extensive vomiting, a secondary disease, such as IBD in Hugo's case, should be considered.

References:

- 1) Brown, B.; Mauldin, G.E.; Armstrong, J.; Moroff, S.D.; Mauldin, G.N. Metabolic and Hormonal Alterations in Cats with Hepatic Lipidosis. *Journal of Veterinary Internal Medicine*. 14(1): 20-6. 2000.
- 2) Ettinger S.J., Feldman E.C. *Textbook of Veterinary Internal Medicine*, Sixth Edition. Elsevier Saunders. 2005. P. 1471-1473.
- 3) Richter, K. Common Feline Hepatopathies. *Tufts Animal Expo* 2002. VIN.
- 4) Center, S.A. Feline Hepatic Lipidosis. *Veterinary Clinics of North America Small Animal Practice*. 35(1): 225-69. 2005.