



ACUTE HEPATOPATHY IN A JUVENILE CANINE

Case Report
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Signalment:

“Oscar”
2-month old MI West Highland White Terrier
BCS 5/9

History:

Oscar was presented for vomiting 10-15 times over a 24 hour period, approximately 2 weeks after he was purchased. Foreign-body or toxin ingestion was considered unlikely, but wild mushrooms grow in the backyard, and he has a history of chewing on acorns. Raccoons and coyotes also frequent the backyard.

Oscar initially presented to the primary veterinarian weak, minimally responsive, and with pale mucous membranes. Bloodwork was analyzed in-house and revealed increased liver enzymes, hypoglycemia, and a mild leukocytosis (see laboratory findings section). An IV catheter was placed, IV fluids were started, and dextrose was administered multiple times with improvement seen in blood glucose afterwards. Radiographs of his abdomen demonstrated a focal area of gas within the intestinal lumen towards the right side. Despite normoglycemia, Oscar was still lethargic and was transferred to VMSG for further care. Oscar was not on any medications at the time. He was vaccinated for canine distemper virus, canine adenovirus-2, parvovirus, and parainfluenza on 9/08. He was also vaccinated for Coronavirus on 10/02/08 and had negative fecal exams on 10/2/08 and 10/4/08.

Clinical Exam:

On presentation, Oscar was quiet but responsive, normothermic, tachycardic (PR 260), and tachypneic (RR 80). His abdomen palpated fluid-filled, and he appeared nauseated during the palpation. There were no significant findings on rectal exam, and both testicles were descended. There was no peripheral lymphadenopathy.

Laboratory Findings:

10/11/09 - Primary veterinarian: ALP 457, ALT 2730, Glucose 12 (improved to 141), WBC 19.3, HCT 37.3%, Plt 442
10/12/09 - (In-house, sample required dilution): ALP 860, ALT 6000-8000, total BILI 1.2, BG 70, BUN 8, CHOL 105-140, Resting Bile Acids 130 - 150. PT out of range, PTT 228s. PCV 40%, TS 4.4g/dl
10/13/08 - (In-house) ALB 2.7, ALP 1184, ALT out of reference range, tBILI 2.1, BUN 3, CREAT <0.2, PHOS 7.2, GLUC 86, TP 3.8, GLOB 1.2. PT and PTT out of reference range. PCV/TS: 32%, TS 4.2g/dl
10/14/08 - PT 26s, PTT 132s
10/15/08 - PT 19s, PTT 119s
10/15/08 – Discharge: CBC: WBC 16.6, HCT 35%, LYMPH 8466, NEUT 7138, EOS 332, BASO 166, PLT 451
Chemistry Panel: AST 164, ALT 2537, ALP 1803, GGT 22, tBILI 2.3, ALB 2.3, GLOB 2.4, BUN 5, CREAT 0.5, PHOS 8.6
10/18/08 - Primary DVM: WBC 20.2, HCT 34.6%, LYMPH 7070, NEUT 9898, MONO 3030, PLT 156 (with clumping)

Diagnostic Imaging:

Abdominal ultrasound findings on presentation: Normal appearing hepatic parenchyma (Fig 1 & 2). Mild mesenteric and sublumbar lymphadenopathy (Fig 3 & 4) with no other apparent abnormalities.

Fig 1-4 (left to right). Static sonographic images of the hepatic parenchyma and abdominal lymph nodes.



Diagnosis:

Acute hepatopathy – The primary rule-outs based on signalment and clinical data were an acute toxic hepatopathy and infectious/inflammatory hepatopathy. Portosystemic shunting as a sole cause was considered unlikely given the extreme elevations in liver enzymes, atypical for this developmental abnormality. Suspicion for early hepatic failure was supported by clinical evidence of hypoglycemia, increased resting bile acids, low-end of normal BUN and cholesterol, and secondary coagulopathy. Oscar's vomiting was thought to be secondary to either acute the hepatopathy and/or directly from toxin ingestion.

Treatment/Management:

Oscar was presented through the Emergency Service on 10/11/09. While in triage, Oscar vomited multiple times and appeared depressed. An abdominal ultrasound was performed (see above findings). Oscar was admitted to the ICU, started on intravenous fluids (0.9% NaCl with dextrose), enrofloxacin, ampicillin, metronidazole, famotidine, and dolasetron.

The following day, Oscar's mentation was still dull, and he had several episodes of hematemesis. He was dehydrated, mucous membranes were paler than at presentation, and ALT was increased and markedly disproportionately higher compared to other the liver values; coagulation times were also markedly elevated. Oscar was continued on his previous treatments with the addition of vitamin-K1 and low dose metoclopramide; fresh frozen plasma was administered, and the owners were updated on his guarded prognosis.

Oscar was hospitalized for the next 3 days in the ICU, and experienced progressive improvement in mentation and resolution of hematemesis. He received two additional fresh frozen plasma transfusions, with an improvement in clotting times seen afterwards. When he began to hold down Pediasure, he was started on Denamarin. His liver parameters remained elevated through hospitalization, though a decrease in severity was noted. At discharge on 10/15/09, he was eating well, on oral medications only, and coagulation times were significantly improved though still mildly outside the reference range.

Post-hospitalization care:

The owners were instructed to monitor for vomiting, coughing, lethargy, and labored or fast breathing. Medications to be continued were enrofloxacin, Clavamox, famotidine, metoclopramide, and Denamarin.

Follow-up:

For the next several days after discharge, Oscar had waxing and waning episodes of lethargy, though overall he progressively improved and began to have the energy level of a puppy again. One week after discharge he was feeling well with complete resolution of his presenting clinical signs, and bloodwork showed gradual improvement in liver values over a period of several weeks.

Discussion:

Hepatopathies in dogs can be divided into categories such as chronic hepatitis (CH), acute hepatitis (AH), acute-toxic insult, metabolic, portal vascular anomalies, and hypoxemic or reactive injury. CH is histologically a disease caused by lymphoplasmacytic inflammation, with granulomatous and eosinophilic inflammatory sub-types less common. Other causes of CH include copper accumulation, lobular dissecting hepatitis, fibrosis/cirrhosis, and occasionally chronic Leptospirosis. With the exception of lobular dissecting hepatitis, the other forms of CH are rare in juveniles. Additionally, with lobular dissecting hepatitis the patient is often refractory to anti-inflammatory treatment, the liver has acute fibrosis, and the disease is usually lethal in several weeks [2], making this disease unlikely in Oscar's case.

A hepatopathy in a juvenile dog with sudden clinicopathologic manifestations and features of disproportionately high hepatocellular leakage enzymes (compared to cholestatic enzymes) like Oscar had would exclude most etiologies of hepatitis other than AH caused by either toxic ingestion, or injury secondary to circulating endotoxin, hypoxia, or trauma [1]. There is a lack of supportive history and clinical markers for sepsis/endotoxemia, hypoxemia, and known trauma making toxin ingestion and infection the remaining differentials.

Most acute toxin injuries occur from either ingestion of naturally occurring organic compounds like aflatoxin (mycotoxin), phalloidine (specifically mushroom toxin), or from heavy metals [8,9]. Bacterial infectious causes of hepatitis are rare, with biliary infectious cholecystitis the more common [7]. Infection in incompletely vaccinated juvenile dogs is most often due to canine adenovirus-1, and as with toxic injury, laboratory and clinical signs relating to hepatic toxicity are similar to what Oscar had. These include markedly increased hepatocellular leakage enzymes, hyperbilirubinemia/icterus, hypoalbuminemia, hypoglycemia, hypocholesterolemia, nausea, and a coagulopathy.

Because the liver is often affected diffusely, radiography can be unrewarding. Ultrasonography can be sensitive in distinguishing between echogenic differences relating to parenchymal changes, though the sonographic diffuse variation is seldom specific for any particular etiology [3]. A liver FNA, needle-biopsy, or wedge biopsy sample may be

helpful in excluding an infectious or malignant cause, though these technique may also be contraindicated if coagulation times are prolonged or if treatment is to likely remain unchanged, as in Oscar's situation.

Treatment is primarily supportive and typically includes vitamin K₁, vitamin E, S-adenosylmethionine, milk thistle, nutritional support, gastric protectants, antiemetics, and fresh-frozen plasma if indicated [4,5,6]. Complete recovery usually occurs as long as overwhelming irreparable hepatocellular damage has not occurred, and treatment is able to be continued until clinical signs resolve.

References:

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