

CASE REPORT #1

SIGNALMENT

Oliver, 2 year old, male/neutered, Wheaton terrier.

HISTORY

Oliver presented to the Internal Medicine service at the Veterinary Emergency Clinic and Referral Centre for a diagnostic evaluation of ascites, hypoproteinemia, and hypoalbuminemia on October 1, 2012. Oliver's history included gradual weight loss over the previous two months and slightly soft stools. Current history included decreased appetite and two episodes of vomiting over the previous three days. The owners reported that Oliver's mentation had not changed and that he was his normal energetic self. His vaccinations were current and he had no travel history. A complete blood count (CBC), serum biochemical profile, pre and post prandial bile acids, urine protein/creatinine ratio, and cytology of abdominal effusion were performed by the referring veterinarian (RDVM). The CBC was normal. The serum biochemical results revealed marked hypoproteinemia (29g/L, N=54-75 g/L), hypoalbuminemia (20g/L, N=31-43 g/L), hypoglobulinemia (13g/L, N=18-39g/L), and mild hypocalcemia (2.15mmol/L, N=2.20-3.00mmol/L). Pre and post prandial bile acids were normal (pre 3.0umol/L, N=<10; post 15.1umol/L, N=<20). The protein/creatinine ratio was normal at 0.1 (normal <0.5). Cytology of the abdominal effusion revealed a transudate.

INITIAL PHYSICAL EXAM

Upon arrival at the VEC, Oliver was bright, alert and responsive. His physical exam revealed: a body weight of 13.7kg, cachexia with a body condition score (BCS) of 1.5/5 (very thin – thin), normal temperature (T) of 38.4C, heart rate (HR) of 114bpm, and a respiratory rate

(RR) of 36bpm. He had a large distended abdomen with a fluid wave, and muffled heart sounds bilaterally.

PROBLEM LIST

Problem list included: cachexia, ascites, muffled heart sounds, hypoalbuminemia, hypoproteinemia, and hypoglobulinemia. Prognosis was guarded.

INITIAL DIAGNOSTICS AND RESULTS

Oliver was admitted to the hospital for a workup that consisted of an abdominal ultrasound, thoracic radiographs, and gastroduodenoscopy. Ultrasound and gastroduodenoscopy were performed by the veterinarian (DACVIM) and assisted by the technician (RVT), and the radiographs were performed by the RVT. The abdominal ultrasound revealed a marked amount of free abdominal fluid. The abdomen was sterilely prepped and abdominocentesis was performed using a 19G angiocath - 1370ml of clear, colourless fluid was removed. The thoracic radiographs revealed moderate pleural effusion with no evidence of mediastinal lymphadenopathy or intrathoracic masses. The heart chambers were within normal size limits. The lateral thorax was clipped and sterilely prepped and thoracocentesis was performed using a 20G IV catheter - 350ml of clear, colourless fluid was removed, sent for cytology and was confirmed to be a transudate.

Prior to gastroduodenoscopy, a peripheral catheter (20G x 1") was placed in the right cephalic vein and intravenous fluids were initiated using a colloid solution (PentaspanTM – 10% pentastarch in 0.9% NaCl) at a rate of 3ml/kg/hr (41ml/hr). Colloids were chosen instead of crystalloids to increase the oncotic pressure and limit extravascular fluid accumulation¹. A plasma transfusion was an alternate choice, but due to cost and the fact that Oscar's albumin was

>1.5g/L, colloids were prescribed instead. Oscar was premedicated with butorphanol 0.2mg/kg IM (2.7mg/0.27ml of 10mg/ml) and induced with propofol 2-4mg/kg IV (27.4-54.8mg/2.7-5.5ml of 10mg/ml) titrated to effect. He was then intubated with a 9.0mm Magill endotracheal tube and maintained on 2% isoflurane and 1.5L/min of oxygen. Oscar's vital parameters were monitored with a SurgivetTM (HR, BP, ECG, SpO₂, and ETCO₂). His heart rate and pulse quality were also monitored by palpation of the femoral artery and via auscultation. The DACVIM then performed endoscopy of his stomach and duodenum, and with the assistance of the RVT, biopsies were collected for histopathology and were submitted to the pathologist. The stomach appeared grossly normal, and the small intestine had pale white nodules and inflamed mucosa. Due to the appearance of the small intestine, the main rule out was dilated lacteals with lymphangiectasia. Oscar's anesthetic lasted 25 minutes and was unremarkable. He maintained a HR of 80-100bpm, a MAP of 80mmHg, normal sinus rhythm on ECG, an SpO₂ of 98% and an ETCO₂ of 35mmHg. His recovery was uneventful. The colloid fluid rate was decreased to 1ml/kg/hr (14ml/hr) and crystalloids (PlasmalyteTM) were added at half maintenance rate of 14ml/hr (maintenance being 50ml/kg/day, 28ml/hr).

Oscar remained in the hospital overnight for post anesthetic monitoring. The working diagnosis was lymphangiectasia, so immunosuppressive therapy was initiated with dexamethasone 0.25mg/kg IV q 24hr (3.43mg, 0.69ml of 5mg/ml) as well as a low fat diet to be offered once Oscar was alert. He had an uneventful night and in the morning he was bright, alert and responsive. His vitals were within normal limits: Blood pressure (BP) 126/71 MAP 87, T=38.4°C, P=88bpm, R=32bpm, and his mucous membranes were pink and moist. He ate well overnight and there had been no vomiting or diarrhea. He had no appreciable pleural effusion on

tFAST and minimal abdominal effusion on aFAST. A blood sample was taken for PCV/TP (40%/4.0g/dL – normal PCV, hypoproteinemic), BG (7.0mmol/L - normal), and electrolytes (Na 145mmol/L, K 3.4mmol/L, Cl 118mmol/L – all within normal ranges). Oscar remained in hospital for the day to wean him off of IV fluid therapy and to ensure that he was eating well. The colloids were discontinued in the morning and the Plasmalyte™ at noon. The pathology results were still pending at this time, but based on bloodwork, bicavital effusion, and endoscopic findings, treatment for lymphangiectasia was initiated. His medication was prepared by the RVT, on orders from the DACVIM: prednisone 1mg/kg PO q12hr (15mg) for 1 month. The owner was given strict instructions to feed a low fat diet only, along with a vitamin supplement containing the fat soluble vitamins A, D, E, and K to be purchased at any drug store. Oscar was to return in one week for follow-up blood work.

LABORATORY RESULTS

Histopathology revealed massive distention of the villus lacteals with utterly convincing lesions of small intestinal lymphangiectasia.

FOLLOWUP EXAMINATIONS

Oscar returned for a recheck 2 weeks later. On physical exam, he was bright, alert and responsive. His physical exam revealed: a body weight of 13.0kg (loss of 0.7kg), still cachexic with a body condition score (BCS) of 1.5/5, normal temperature of 38.5C, heart rate of 126bpm, and a respiratory rate of 24bpm, heart and lung sounds normal on auscultation. He had a distended abdomen with a fluid wave. The owner reported polyuria, polydipsia, and polyphagia due to the prednisone. They also reported that he had developed diarrhea 3 days prior, but had not experienced any vomiting. Blood was collected by the RVT and sent for albumin (19g/L)

and total protein (34g/L). An abdominocentesis was performed and 200ml of clear, colourless fluid was removed. Oscar was discharged with the same instructions as prior, plus a prescription of tylocine 200mg POq 12h for 2 weeks to treat the diarrhea. After 14 days, he returned for a follow-up exam. Vitals and weight were unchanged from the previous visit and the owner reported that he was still bright and alert and his appetite was good. His diarrhea had resolved. Blood was collected by the RVT for albumin (21g/L) and protein (34g/L)

Oscar returned for another recheck 11 days later. On physical exam, he was quiet but alert and responsive. His physical exam revealed: a body weight of 12.5kg (loss of 0.5kg) and he was still cachexic with a body condition score (BCS) of 1-1.5/5. His temperature was normal at 38.1C, he had a heart rate of 120bpm, and was panting with muffled heart and lung sounds on auscultation. He had a distended abdomen with a fluid wave. His owner reported that Oscar's appetite was poor and he had progressively become less active over the previous week. tFAST and aFAST ultrasounds were performed by the DACVIM and bicavital effusion was found. Abdominocentesis was performed and yielded 680ml of clear, colourless fluid. Thoracocentesis yielded 360ml of the same. Blood was collected by the RVT for CBC and biochemical profile which revealed hypoproteinemia (TP 36g/L), hypoalbuminemia (22g/L), hypoglobulinemia (14g/L) and hypocalcemia (2.13g/L), no other abnormalities were noted on bloodwork. His mentation improved significantly after thoracocentesis was performed and he ate a full meal of canned low fat dog food in the hospital, so the owners elected to take him home with a prescription of furosemide 1mg/kg (15mg) PO q24hours, in addition to his current treatment regime, to try to minimize the effusion. His prognosis was grave due to his poor response to

treatment. Five days later, Oscar was lethargic, inappetant, and dyspneic at home so the owners took him in to their family veterinarian and humane euthanasia was performed.

<u>TEST</u>	<u>RDVM bloodwork Initial Presentation</u>	<u>OCT 16/12 Recheck Evaluation</u>	<u>NOV 1/12 Recheck Evaluation</u>	<u>NOV 12/12 Recheck Evaluation</u>
Albumin (31-43g/L)	20g/L	19g/L	21g/L	22g/L
Total Protein (54-75g/L)	29g/L	34g/L	34g/L	36g/L
Globulin (18-39g/L)	13g/L			14g/L
Calcium (2.2-3.0mmol/L)	2.15mmol/L			2.13mmol/L
Weight (kg)	13.7	13.0	13.0	12.5

DISCUSSION OF CASE

The main rule outs for hypoalbuminemia are hepatic insufficiency, protein losing nephropathy (PLN), and protein losing enteropathy (PLE).² PLE is a disease associated with excessive loss of protein or other protein-containing tissues into the gastrointestinal tract.³ Intestinal lymphangiectasia is one cause of PLE. There are two types of lymphangiectasia – primary and secondary. Primary lymphangiectasia is a progressive congenital disorder that is usually limited to the intestine. It is most commonly seen in small terrier breeds (including the Wheaton Terrier), and the Norwegian Lundehund. Histologically, it is characterized by a distension of the submucosal lymphatic vessels and dilation of the lacteals in the villi. These affected lymphatics leak protein-rich lymph into the intestinal tract causing hypoproteinemia⁴, and can result in an inability to maintain fluid balances in the interstitial spaces, resulting in ascites, subcutaneous edema, and even pleural effusion.⁵ Secondary lymphangiectasia is caused

by intestinal lymphatic obstruction due to: inflammation, neoplasia, fibrosis, blockage of the thoracic duct, or right heart failure due to congestive heart failure or cardiac tamponade.⁶

Oscar's initial workup was performed to differentiate the cause of his hypoproteinemia, weight loss, pleural effusion, and ascites. Elimination of liver disease was based on a normal bile acid and liver function tests. Protein-losing nephropathy was eliminated based on kidney function and the urine protein/creatinine ratio, which were normal. The radiographs eliminated heart failure and any mass lesions obstructing the thoracic duct. The abdominal and pleural fluids were pure transudate, and with right sided heart failure, the portal hypertension would have caused a modified transudate.⁷ Endoscopy findings, both grossly and histologically, were consistent with primary duodenal lymphangiectasia.

Secondary lymphangiectasia can be treated by managing or correcting the underlying cause of the disease, such as pericardectomy to correct cardiac tamponade. With primary lymphangiectasia, the goal is to control the disease by decreasing the enteric loss of protein, resolving the intestinal or lymphatic inflammation, and controlling the effusion. A diet that is low in fat and dense in calories is usually recommended. Theoretically, a low fat diet reduces distension of intestinal lymphatics, which are mainly required for transportation of dietary fat. Most low fat diets are also low in nutrition, so medium chain triglycerides are usually added by means of a fat-soluble vitamin supplement. Glucocorticoid therapy is also given to inhibit inflammation and promote enterocyte function, usually starting with an immunosuppressive dose of prednisone or prednisolone at 1-2mg/kg PO q 12-24hr, followed by a gradually decreasing regime. Ideally, clinical signs will disappear and the animal can go into remission for months to

years, but the prognosis is poor due to the unpredictability of the response to treatment, and patients eventually succumb to severe malnutrition, effusion and diarrhea.⁸

¹ William, David A., Malabsorption, Small Intestinal Bacterial Overgrowth, and Protein-Losing Enteropathy, in **Strombeck's Small Animal Gastroenterology, Third Edition**, Guilford, W. Grant, Center, Sharon A., Strombeck, Donald R., Williams, David A., Meyer, Denny J., W.B. Saunders Company, Philadelphia, PA., 1996
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² Nelson, Richard W. and Couto, C. Guillermo, Chapter 29, in **Small Animal Internal Medicine, Third Edition**, Mosby, Inc, 2003 Pg366.

³ William, David A., Malabsorption, Small Intestinal Bacterial Overgrowth, and Protein-Losing Enteropathy, in **Strombeck's Small Animal Gastroenterology, Third Edition**, Guilford, W. Grant, Center, Sharon A., Strombeck, Donald R., Williams, David A., Meyer, Denny J., W.B. Saunders Company, Philadelphia, PA., 1996
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⁴ Hall, Edward J. and German, Alexander J., Section V111, Chapter 222, Diseases of the Small Intestine: Lymphangiectasia, in **Textbook of Veterinary Internal Medicine; Diseases of the Dog and Cat, Sixth Edition; Volume II**, Ettinger, Stephen J and Feldman, Edward C., Elsevier Saunders, St. Louis, Missouri, 2005 Pg. 1373

⁵ William, David A., Malabsorption, Small Intestinal Bacterial Overgrowth, and Protein-Losing Enteropathy, in **Strombeck's Small Animal Gastroenterology, Third Edition**, Guilford, W. Grant, Center, Sharon A., Strombeck, Donald R., Williams, David A., Meyer, Denny J., W.B. Saunders Company, Philadelphia, PA., 1996
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⁶ Hall, Edward J. and German, Alexander J., Section V111, Chapter 222, Diseases of the Small Intestine: Lymphangiectasia, in **Textbook of Veterinary Internal Medicine; Diseases of the Dog and Cat, Sixth Edition; Volume II**, Ettinger, Stephen J and Feldman, Edward C., Elsevier Saunders, St. Louis, Missouri, 2005 Pg. 1373

⁷ Hall, Edward J. and German, Alexander J., Chapter 43, Abdominal distention, Ascites, and Peritonitis, in **Textbook of Veterinary Internal Medicine; Diseases of the Dog and Cat, Sixth Edition; Volume I**, Ettinger, Stephen J and Feldman, Edward C., Elsevier Saunders, St. Louis, Missouri, 2005 Pg. 150

⁸ Hall, Edward J. and German, Alexander J., Section V111, Chapter 222, Diseases of the Small Intestine: Lymphangiectasia, in **Textbook of Veterinary Internal Medicine; Diseases of the Dog and Cat, Sixth Edition; Volume II**, Ettinger, Stephen J and Feldman, Edward C., Elsevier Saunders, St. Louis, Missouri, 2005 Pg. 1374-1375