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CASE STUDY
VOLUME 15/ISSUE 6

"Chewie"

Signalment:

"Chewie", a 3year old, 5.0kg, neutered male Yorkshire Terrier.

History:

Chewie had exhibited vomiting and diarrhea for the past four days, and had become progressively more lethargic. The day prior to presentation the vomiting and diarrhea subsided, although the patient continued to retch multiple times during the day. On the day of presentation, the patient would try to drink and would vomit water within a few minutes of consuming it. The client had taken Chewie to their primary care veterinarian who had performed abdominal x-rays (Figure 1), blood work (Table 1), and had referred the patient to IndyVet for further care. The client asserted that Chewie has a tendency to get into trash. The patient was up to date on vaccines and was on a monthly heartworm preventative medication.

Table 1

CBC	June 27, 2015	
Test	**Results**	**Reference Range**
RBC	8.21 M/UL	5.50-8.50 M/µL
HCT	46.80%	37 - 55.0%
HGB	**19.2 g/dL**	12.0 - 18.0 g/dL
MCV	**57.0 fL**	60 - 77 fL
MCHC	**41.0 pg**	18.5-30.0 pg
% RETIC	0.40%	
RETIC	31.2 K/uL	10.0 - 110.0 K/uL
WBC	**54.9 K/uL**	5.50 - 16.90 K/uL
% NEU	**92.9% 50.4 K/ul**	2.22 - 12.00 K/uL
% LYM	4.8% 2.6 K/ul	0.50 - 4.90 K/uL
% MONO	2.1% 1.1 K/ul	0.30 - 2.00 K/uL
% EOS	0%	0.10 - 1.49 K/uL
% BASO	0%	0.0 - 0.10 K/uL
PLT	188 K/uL	148 - 484 K/uL

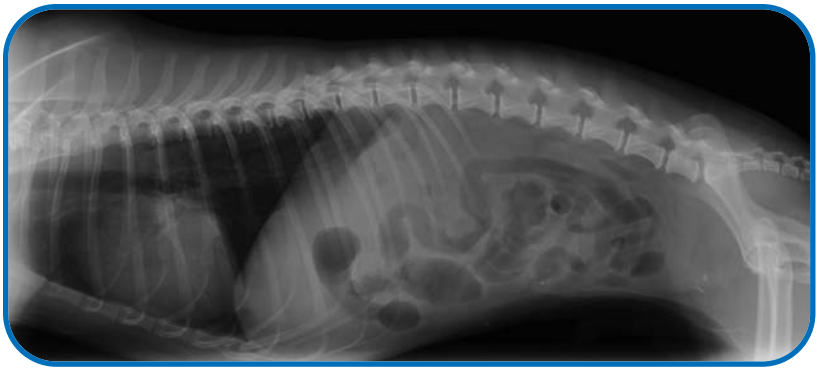
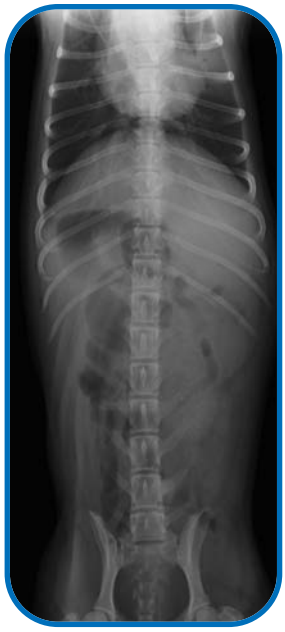


Figure 1



What abnormalities do you see?

Physical Examination:

The patient arrived to IndyVet about 6 hours after being seen by the primary care veterinarian upon getting substantially worse clinically. On physical examination the patient was laterally recumbent. The temperature was 98.2 F, respiration rate was 50 and the patient had tachycardia (160) with weak synchronous matching pulses and a capillary refill time of 4 seconds. Chewie was estimated to be 12% dehydrated, and auscultation suggested mild harsh lung sounds to the left chest. Abdominal palpation was soft and non-painful, however rectal exam revealed hematechezia. The patient was very weak and was neurologically obtunded. The remainder of the physical exam was unremarkable. The patient was quickly admitted to the hospital whereupon a blood pressure, CBC, serum biochemical panel, CPLI, urinalysis, baseline cortisol (Table 2), re-check abdominal x-rays (Figure 2), chest x-rays (Figure 2), and FAST scan were performed.

Table 2

CBC	June 27, 2015	
Test	**Results**	**Reference Range**
RBC	6.72 M/ul	5.50-8.50 M/µL
HCT	38%	37 - 55%
HGB	15.5 g/dl	12.0 - 18.0 g/dL
MCV	56.8 fl	60 - 77 fL
MCHC	**40.6 g/dl**	18.5-30.0 pg
% RETIC	0.30%	
RETIC	16.8 K/ul	10.0 - 110.0 K/uL
WBC	**40.37 K/ul**	5.50 - 16.90 K/uL
% NEU	**83.6 % / 33.8 K/ul**	2.22 - 12.00 K/uL
% BANDS	**2% / 0.80 K/ul**	
% LYM	11.6% / 4.68 K/ul	0.50 - 4.90 K/uL
% MONO	2.7% / 1.1 K/ul	0.30 - 2.00 K/uL
% EOS	0.00%	0.10 - 1.49 K/uL
% BASO	0.1% / 0.03 K/ul	0.0 - 0.10 K/uL
PLT	189 K/ul	148 - 484 K/ul

Table 2 [Continued]		
Serum Biochemical Profile		**Reference Range**
GLU	120 mg/dl	74 - 143 mg/dL
BUN	**100 mg/dl**	7 - 27 mg/dL
CREAT	1.3 mg/dl	0.5 - 1.8 mg.dL
PHOS	**8.9 mg/dl**	2.5 - 6.8 mg/dL
CA	7.9 mg/dl	7.9 - 12.0 mg/dL
TP	6.6 g/dl	5.2 - 8.2 g/dL
ALB	3.4 g/dl	2.3 - 4.0 g/dL
GLOB	3.2 g/dl	2.5 - 4.5 g/dL
ALT	31 U/L	10 - 100 U/L
ALKP	106 U/L	23 - 212 U/L
GGT	5 U/L	0 - 7 U/L
TBIL	0.5 mg/dl	0.0 - 0.9 mg/dL
CHOL	174 mg/dl	110 - 320 mg/dL
AMYL	**438 U/L**	500 - 1500 U/L
LIPA	423 U/L	200 - 1800 U/L
Na	**110 mmol/L**	144 - 160 mmol/L
K	3.9 mmol/L	3.5 - 5.8 mmol/L
Cl	**75 mmol/L**	109 - 122 mmol/L
cPLi	Normal	Normal
Baseline Cortisol	> 10 ug/dl	>5 ug/dl
Blood Pressure	74/50	

Urinalysis	**June 27, 2015**	**Cystocentesis Collection**	
Color	Yellow	pH	5
Glucose	Negative	Protein	30 mg/dl
Bili	1 mg/dl	Urobili	Normal
Ketone	Negative	Leukocytes	Negative
Sp. Gravity	1.033	Blood	10 / uL
Bacteria	Negative	WBCs	3 - 5 / HPF
Crystals	None	RBCs	20 - 30 / HPF

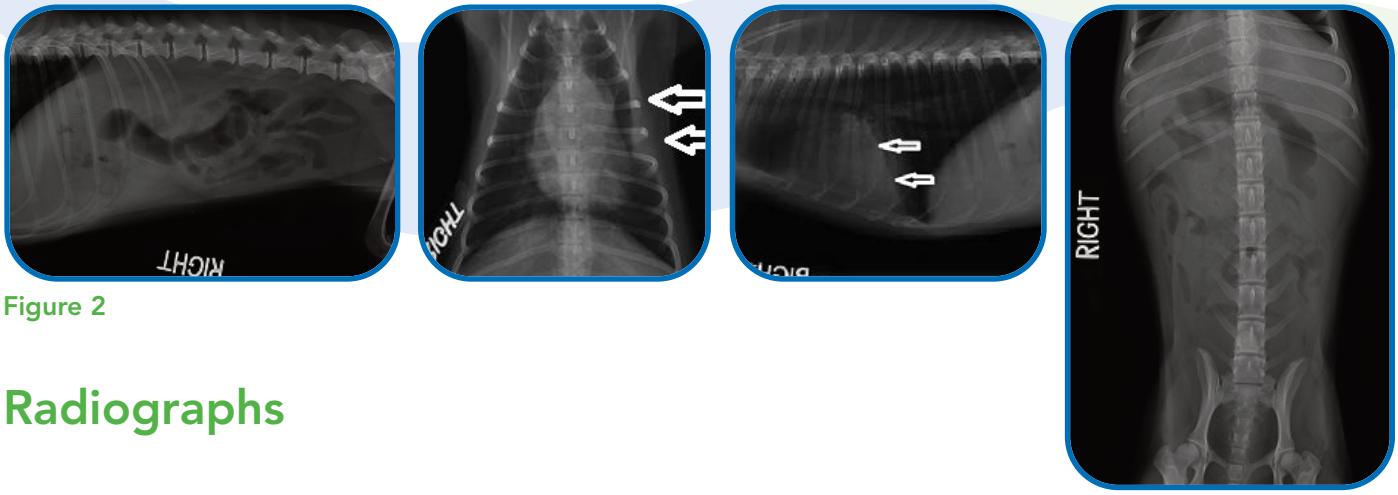


Figure 2

Radiographs

Interpretation:

The abdominal radiographs are unremarkable. On the thoracic radiographs there is a distinct soft tissue density demarcation on the right lateral projection that angles across the cardiac silhouette in conjunction with retraction of the pulmonary parenchyma from the thoracic wall at the level of the 4th to 6th ribs on the VD projection, consistent with consolidation of the posterior portion of the left cranial lung lobe (see arrows). Impression: likely aspiration pneumonia.

Fast Scan:

A four quadrant FAST scan revealed no free fluid.

Treatment:

An IV catheter was placed and a 100 ml bolus of LRS fluids was given followed by a twice maintenance rate of fluids at 20cc/hr. Maripotent (1mg/kg), famotidine (1mg/kg), metronidazole (10mg/kg), and ampicillin (22 mg/kg) were given intravenously and the patient was warmed with a Bair hugger. Orders were given to prepare for an abdominal ultrasound.

Abdominal Ultrasound:

Unremarkable structures observed during abdominal ultrasound included the liver, spleen, adrenals, kidneys, pancreas, bladder and sub-lumbar lymph nodes. The gall bladder was markedly distended with normal anechoic bile. The stomach was not distended and showed normal rugal folds, wall thickness, and motility. Multiple loops of small intestine were moderately distended with fluid and ingesta and the colon was moderately distended with a large amount of fluid fecal material. Multiple mesenteric lymph nodes were moderately enlarged. The ileum was intermittently seen to reversibly involute into the colon (Figure 3), and a thickened piece of bowel was seen just oral to the affected ileum which could be consistent with a foreign body, but could not be substantiated. Barium administration was recommended for further evaluation for a potential foreign body.

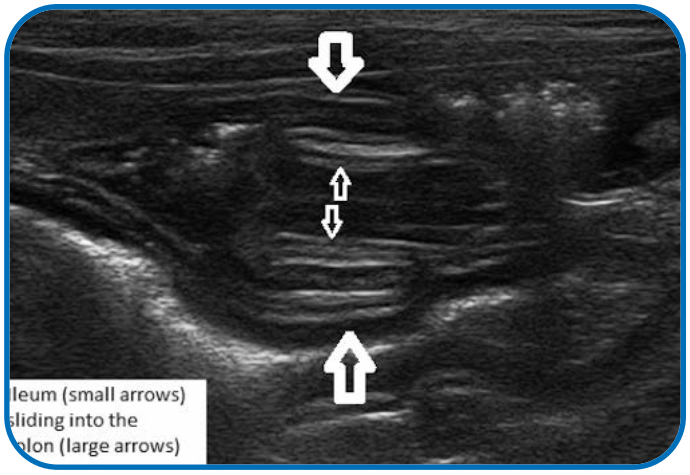
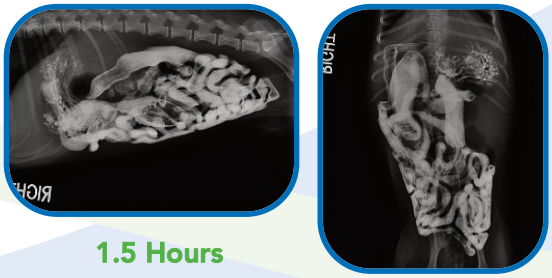


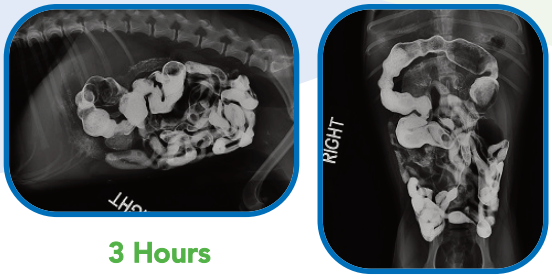
Figure 3

Barium Study:

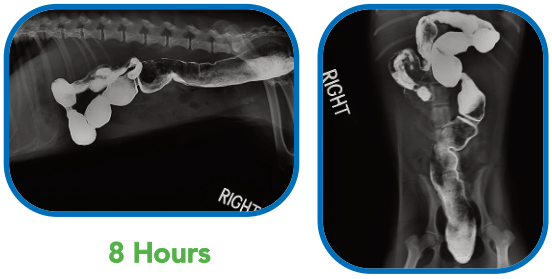
20cc/kg of barium was administered and a non-standard modified barium series was performed with lateral and VD views taken at 1.5 hours, 3 hours, and 8 hours due to the primary concern for an occult intestinal foreign body.



1.5 Hours



3 Hours



8 Hours

What is your diagnosis?

How would you treat this patient now?

Treatment:

Fenbendazole (50mg/kg PO SID), and sucralfate (0.5gm PO, TID) were added to the medical regimen and the patient was carefully re-hydrated over 48 hours monitoring the electrolyes every 12 hours (Table 3). Chewie was offered small amounts of food which he ate. The stools remained very soft. Vomiting subsided for 48 hours, but Chewie started regurgitating again the following day and remained very lethargic despite his temperature, pulses, respirations, and blood pressure returning to normal and being appropriately hydrated. A re-check CBC, biochemical profile (Table 3), and abdominal ultrasound was ordered.

Table 3

	3:20 AM	1:45 PM	9:46 AM		
CBC	**June 28, 2015**	**June 28, 2015**	**June 29, 2015**	**June 30, 2015**	
Test				**Results**	**Reference Range**
RBC				4.87 M/ul	5.50-8.50M/uL
HCT				35%	37 - 55%
HGB				**11.1 g/dl**	12.0 -18.0 g/dL
MCV				61.8 fl	60 - 77 fL
MCH				22.8 pg	21.2 - 25.9 pg
MCHC				36.9 g/dl	18.5 - 30.0 pg
% RETIC				0.30%	
RETIC				7.3 K/ul	10.0 - 110.0 K/uL
WBC				**31.47 K/ul**	5.50 - 16.90 K/uL
% NEU				**83. % / 26.1 K/ul**	2.22 - 12.00 K/uL
% LYM				10% / 3.15 K/ul	0.50 - 4.90 K/uL
% MONO				**6.6% / 2.08K/ul**	0.30 - 2.00 K/uL
% EOS				0.3% / 0.09 K/ul	0.10 - 1.49 K/uL
% BASO				0.1% / 0.03 K/ul	0.0 - 0.10 K/uL
PLT				177 K/ul	148 - 484 K/ul
Serum Biochemical Profile					**Reference Range**
GLU				130 mg/dl	74 - 143 mg/dL
BUN				**3 mg/dl**	7 - 27 mg/dL
CREAT				**0.3 mg/dl**	0.5 - 1.8 mg.dL
PHOS				**1.1 mg/dl**	2.5 - 6.8 mg/dL
CA				**7.7 mg/dl**	7.9 - 12.0 mg/dL
TP				**5.0 g/dl**	5.2 - 8.2 g/dL
ALB				2.3 g/dl	2.3 - 4.0 g/dL
GLOB				2.7 g/dl	2.5 - 4.5 g/dL
ALT				45 U/L	10 - 100 U/L
ALKP				50 U/L	23 - 212 U/L
GGT				1 U/L	0 - 7 U/L
TBIL				0.1 mg/dl	0.0 - 0.9 mg/dL
CHOL				102 mg/dl	110 - 320 mg/dL
AMYL				697 U/L	500 - 1500 U/L
LIPA				443 U/L	200 - 1800 U/L
Na	**116**	**123**	**138**	**142 mmol/L**	144 - 160 mmol/L
K	**3.2**	**3**	**3.1**	3.5 mmol/L	3.5 - 5.8 mmol/L
Cl	**83**	**87**	**97**	**103 mmol/L**	109 - 122 mmol/L

Abdominal Ultrasound:

There was segmental dilation of the small intestines. A hyperechoic structure with distal acoustic shadowing was noted within one intestinal segment in the right lateral abdomen (Figure 4). There was dilation of intestinal segment oral to the structure, and un-dilated intestinal segments aboral to the structure suggesting intestinal foreign body.

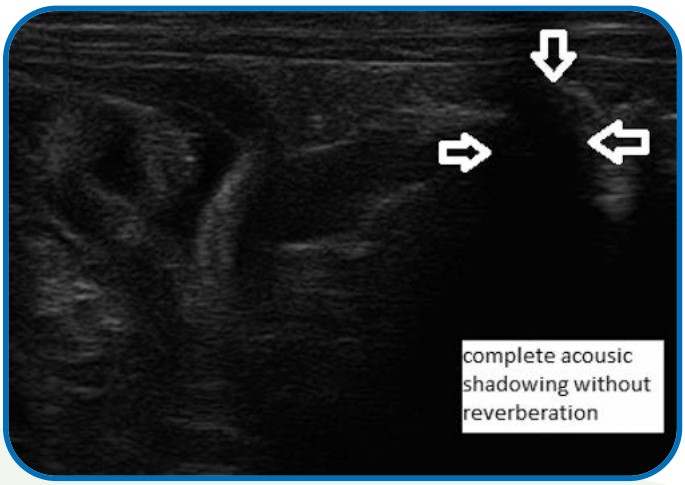


Figure 4

Surgery:

Exploratory laparotomy revealed a peach pit at the junction of the jejunum and ileum. The foreign body was removed routinely. Chewie went home 36 hours later and recuperated uneventfully at home.

Discussion:

This case is an interesting case of an occult foreign body that did not live up to textbook expectations on imaging or laboratory values creating a diagnostic and therapeutic challenge.

Upon admission the patient was laterally recumbent, poorly responsive and neurologically obtunded, likely due to the electrolyte abnormalities secondary to vomiting and diarrhea, or profound dehydration and shock. Clinical signs of hyponatremia are more associated with how rapidly sodium concentrations change than the severity of the loss. Regardless, when sodium levels fall below 120 mEq/L, or decrease at a rate greater than 0.5 mEq/L/hr, water intoxication can occur if the brain's defense mechanisms are acutely overwhelmed. With chronic sodium loss, brain volume can be adjusted toward normal by loss of potassium and organic osmolytes from neurons preventing water toxicity. Clinical signs that may be seen with severe acute hyponatremia occurring over a 24-36 hour period include lethargy, nausea, vomiting, weakness, incoordination, seizure, coma, and death. In Chewie's case neurologic symptoms were present and rapidly getting worse, although the quick response to fluid resuscitation suggested that dehydration and shock were more responsible for his clinical appearance than was the hyponatremia.

The two main goals in treating symptomatic hyponatremia are to manage the underlying disease creating the disturbance, and to increase serum sodium concentrations to restore normal plasma osmolality at a rate not to exceed 10 – 12 mEq/L/hr. As hyponatremia is corrected, the potassium and organic osmolytes lost to the brain cells as a protective response must be restored to the cell to maintain the osmolality of the cell. If hyponatremia is corrected too rapidly, the replacement of these solutes cannot keep pace with the increased sodium concentration from replacement therapy leading to brain dehydration and injury called osmotic demyelinization or myelinolysis. The lesion of myelinolysis occurs several days after correction of hyponatremia and consists of myelin loss and injury to oligodendroglial cells in the pons, thalamus, sub-cortical white matter, and cerebellum and can be detected with MRI. 0.9% sodium chloride or lactated ringers crystalloid fluids (LRS) should be used to correct severe hyponatremia. In the case presented, LRS was selected due to its lower concentration of sodium than normal saline (130 mEq/L versus 154 mEq/L) which would allow a faster and larger volume of fluids to be safely administered to initially manage severe dehydration.

Changes in chloride and sodium concentrations are proportional when the changes are due to increases or decreases in free water. Chloride can be "corrected" for changes in sodium concentration with the formula Cl (corrected) = Cl (measured) x Na (normal)/ Na (measured). In the case presented the corrected chloride was less than the

low normal value for chloride (109) indicating the hypochloremia seen in this patient was in excess of what would be expected from associated sodium losses. This finding was thought to be due to the higher concentration of chloride lost in vomited gastric fluid than fluid lost from concurrent diarrhea. Although vomiting of stomach contents is the most common reason, other disorders associated with low corrected chloride values can include therapy with thiazide or loop diuretics, hyperadrenocorticism, chronic respiratory acidosis, lipemia, and excessive sodium bicarbonate therapy.

The initial radiographs taken by the referring veterinarian did not suggest foreign body or intestinal obstruction, but did show consolidation to the caudal aspect of the left cranial lung lobe (did you see it?) This finding was further substantiated on the subsequent chest films at IndyVet suggesting aspiration pneumonia as the most likely cause for the leukocytosis and left shift seen on the initial CBC, which is an unusual finding in a simple intestinal foreign body case that has not perforated the bowel.

The elevated BUN with a normal creatinine on this extremely dehydrated patient was suggestive for dehydration or urea recycling from GI blood loss, however resolution of the azotemia prior to correction of the foreign body suggested dehydration more than GI bleed as the cause. Despite close scrutiny with ultrasound, foreign body could not be initially documented. The sliding intussusception did suggest severe gastro-intestinal irritation, and the ileum was thickened proximal to the region that was sliding in and out of the colon. It was thought the patient may have had severe enteritis that was causing a sliding intussusception leading to the clinical signs. Barium was administered to help document if an occult foreign body was present, and secondarily as a therapeutic measure to soothe the GI tract to help medically prevent an irreversible intussusception. The barium series, although not done at standard times, clearly did not show evidence of a foreign body. Had the series been done standardly with views taken at 15, 30, 60, 90, and 120 minutes with follow up films at 4 and 8 hours, the foreign body may have been detected, but this is speculation. Regardless, the facts presented shows how a barium series can miss documenting a foreign body when it is not completely, or nearly completely occluding the intestine. Fortunately a follow up ultrasound identified the foreign body and it was surgically remedied.

Follow up examination found Chewie to exhibit no clinical signs suggestive of myelinolysis. Chewie's owners were so appreciative of everyone's efforts on Chewie's behalf that they brought the IndyVet staff a gift. Peach pie. Irony knows no bounds…..

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CGM can also be used in hospital for new DKAs to avoid over or under dosing insulin in critical patients. In hydrated patients, it can reduce the number of sticks and may remove the need for jugular catheters for sampling in some patients. This monitoring is becoming standard of care for human diabetics and will now make managing veterinary diabetic patients easier as well.

IndyVet now offers this cutting edge technology for diabetic patients.

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