

Inflammatory Bowel Disease in a Sandcat (*Felis margarita*)

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Case report

A wild-born adult male Sandcat of about 10 years old presented with severe weight loss and muscle atrophy despite a very good appetite. Its weight had dropped from 2.9kg in the previous year to 1.6kg, a reduction of 45%. Severe generalized muscle atrophy was noticed and the animal was dehydrated. On only one occasion was diarrhea and vomiting was seen.

Blood and urine was collected under anaesthesia with isoflurane. A urine dipstick gave the following results: pH 5, protein 2+, leucocytes max and Hb 2+; sediment was unremarkable. The blood values were in normal range (see table). An ultrasound of the abdomen did not reveal any abnormalities. A faecal sample did not show any parasites.

Supportive treatment was started with Ringers lactate, aminoacids and amoxicillin. The animal was dewormed with ivermectine and anabolica and vitamin B complex was injected. The amount of food was increased and divided over 4 meals a day. All food was readily consumed. The diet itself was not changed and still consisted of fresh rats from our breeding facility, chicken (with bone) and guinea fowl.

The animal readily gained weight and in 10 days time went from 1.6kg to 2kg again. After three weeks of amoxicillin the treatment was finished and the feeding frequency was gradually reduced to three and later two meals a day as it had been before. After 4 weeks however the weight had dropped again to 1.7kg.

Meals were again given more frequently and probiotics and vitamin powder was added to the food, but the animal kept gradually losing weight and muscle.

Since the blood values did not show an obvious infectious picture, kidney and liver enzymes values were normal, urine samples had normalized after the amoxicillin treatment, no tumors were found and no other symptoms apart from weight loss were seen, a malabsorption syndrome was suspected.

Because of the poor condition of the animal it was decided not to anesthetize the animal to perform a laparotomy and take intestinal biopsies but to try out treatment with a new course of amoxicillin this time combined with prednisolone. The food was still divided over 4 times a day and vitamins and probiotics added. After an initial positive reaction on the treatment with obvious weight gain the animal started losing weight again and finally succumbed about 8 months after it first was presented.

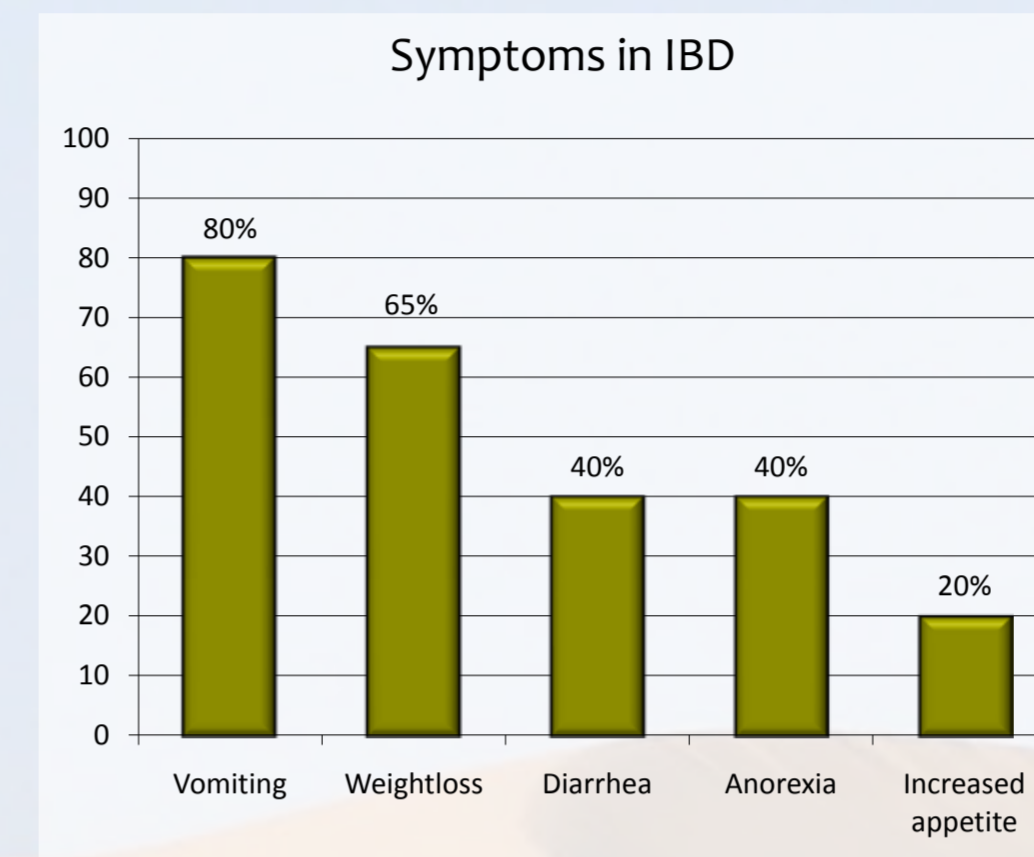
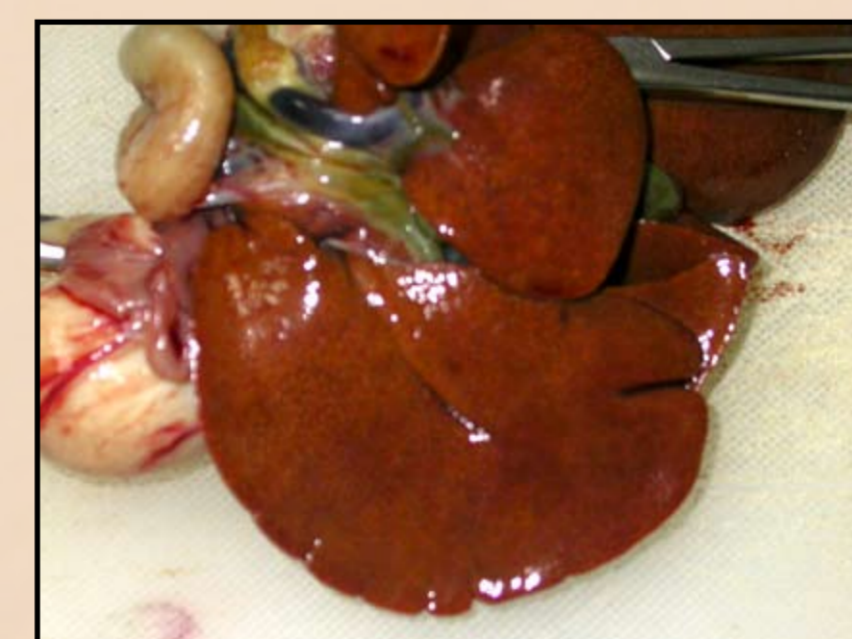
Because of the fluffy coat and the good appetite, and because no vomiting or diarrhea were seen, the keepers only noticed that something was wrong, when the animal had already lost a lot of weight. Therefore therapy might have been initiated too late. No food trial was started because the animal was already in a severely poor condition. Trying out a commercial food to which these animals are not used, and noticing the positive first response to treatment, changing the source of food seemed to be potentially more detrimental to its health. In retrospect, since the diagnosis was only made post mortem, metronidazole and a higher dosage of prednisolone might have been indicated.

	10-Apr-07	15-Dec-07	Reference values ISIS	
Biochemistry				
Total protein	73.1	70.1	51-80	g/l
Albumin	29.6	27.8	26-45	g/l
Globulin	43.5	42.3	19-40	g/l
A:G ratio	0.68	0.66		
Creatinine	106	81	71-150	µmol/l
Urea	13.2	14.4	5.71-14.99	mmol/l
Glucose	5.41	5.34	4.82-11.77	mmol/l
CK	409	3611	30-1967	U/l
AST (GOT)	48.8	117.2	17-121	U/l
ALT (GPT)	81.2	153.1	30-335	U/l
ALP	46	43	5-234	U/l
LDH	147		27-692	U/l
Calcium	2.14	2.24	2-2.9	mmol/l
Phosphate	1.67	1.25	0.97-2.55	mmol/l
Haematology				
Hb	130		87-148	g/l
PCV	0.37		0.26-0.46	
Rbc	9.26		5.6-13.5	x10 ¹² /l
Wbc	3.3		3.6-20.2	x10 ⁹ /l
Neutrophils %	49.5	91		%
N abs count	1.63			x10 ⁹ /l
Lymphocytes %	40.6	7		%
L abs count	1.34		0.31-5.54	x10 ⁹ /l
Monocytes %	2	2		%
M abs count	0.07		0.038-0.93	x10 ⁹ /l
Eosinophils %	8	0		%
E abs count	0.26		0.038-1.21	x10 ⁹ /l
Platelets	350		318-454	x10 ⁹ /l

Post mortem and histopathology

On post mortem the animal showed severe jaundice and a swollen yellow liver. The bladder was filled with thick urine that looked like bile.

On histopathology the following observations were made. In the kidneys round nuclear interstitial infiltrates were seen with in some areas at the change from cortex to medulla a loss of tubuli and fibrosis going in the direction of the pyelum. The spleen had 'depleted' lymphfollicles, there were no obvious sheathed arteries with pals and the red pulpa only had a small amount of red blood cells. The liver was very irregular with severe centrolobular degeneration with foamy hepatocytes, hepatocytes with accumulation of bile and activated macrophages.



Inflammatory Bowel Disease (IBD) in domestic cats

IBD is a well recognized cause of vomiting, weight loss and diarrhea in domestic cats. The term describes a group of chronic infiltrative diseases affecting the lamina propria. Clinical signs vary depending on which parts of the GI tract are affected as well as the severity and type of inflammation (Taboada).

Etiology

The etiology of IBD is unknown but genetics, the environment and the immune response to chronic stimulation from various antigens likely play a role (Taboada). Almost certainly it is related to more than one aetiological factor acting in concert. IBD is essentially a diagnosis of exclusion, i.e., demonstration of certain intestinal inflammatory changes in the intestine of cats with chronic gastrointestinal signs in the absence of any recognisable specific underlying cause (Gruffydd-Jones). IBD usually is most common in middle age to older cats but any age can be affected (Taboada).

Clinical signs

Chronic intermittent vomiting (80% of cases) with or without weight loss (65%) and diarrhea (40%) are the most common clinical signs. Anorexia is seen in 40% of the affected cats but 20% will show an increased appetite. Weight loss may be the only sign in some cats (Taboada).

Diagnosis

Since IBD can mimic many other diseases a thorough evaluation is important. Common differentials include hyperthyroidism, liver disease, intestinal lymphosarcoma, giardiasis, cryptosporidiosis, heartworm, food intolerance, food allergies. The diagnostic workup should include CBC, chemistry panel, urinalysis, T4, X-rays to rule out other causes of the clinical signs observed. The CBC is usually normal, liver enzymes might be mildly increased. Intestinal biopsy is the most important diagnostic test. Endoscopy is useful for biopsy in most cases. Lymphocytic enteritis or enterocolitis is the most common form (Taboada).

Treatment

The mainstays of treatment for IBD are:

- dietary management
- immunosuppressives
- antibiotics

The traditional approach to dietary management is to feed a single source of a novel protein. A newer approach has been based on feeding hydrolysed proteins in which the molecular size of the proteins is minimised to reduce antigenicity.

Prednisolone is the most popular initial choice as an immunosuppressive agent, generally starting with a dosage of 2-4 mg/kg daily. If a good response is achieved this is gradually reduced over a period of generally six to 12 weeks. Alternative immunosuppressive drugs may be used but are generally reserved for refractory cases.

Metronidazole is the antibiotic used most frequently. Often it is reserved for cases which fail to respond to initial treatment with dietary management and prednisolone, or when the IBD appears to be severe. It is questionable whether bacterial overgrowth occurs in IBD but metronidazole may modify the gut flora with potentially beneficial effects. An immunosuppressive action for metronidazole has also been suggested as providing an indication for its use. Other treatments such as probiotics are sometimes used but there are no reports of controlled studies to confirm their value (Gruffydd-Jones).

Prognosis

Little reliable information is available about the expected response to treatment and prognosis for IBD. There are three main reasons why cases fail to respond to routine treatment for IBD: the diagnosis is incorrect, there are complicating factors, the IBD is refractory to the treatment given (Gruffydd-Jones).

References

- Gruffydd-Jones, T., 2006: Inflammatory Bowel disease - Current concepts in Aetiology and Control. Proceedings of Hill's European Symposium on Advances in Feline Medicine, pp 16-21. Available from www.ivis.org.
- Taboada, J., 2005: Inflammatory Bowel Disease. Proceedings of the North American Veterinary Conference, pp 371-372. Available from www.ivis.org

