Successful medical management of asymptomatic feline triaditis: cholangiohepatitispancreatitis-inflammatory bowel disease (IBD)

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Abstract

A neutered male cat was presented to the Milford Veterinary Clinic for periodic health check and annual vaccination. The overweight adult cat had no apparent health issues. However, abnormally high values of several hepatic function blood parameters raised serious clinical concerns. Abdominal ultrasound profiles suggested concurrent cholangiohepatitis, chronic pancreatitis and inflammatory bowel disease (IBD), "triaditis". Dietary treatment regimen with nutraceuticals were advised to the owner.

Keywords: Triaditis, Abdominal ultrasound, Nutraceuticals, Cat

Cat specific cholangitis, pancreatitis, and inflammatory bowel disease (IBD), empirically named "triaditis", of presumptive shared etiology, remains to be clearly deciphered (Marks, 2013; Simpson, 2015). Whereas the clinical signs of cholangitis predominate, pancreatitis and IBD are the major contributors (Fragkou et al., 2016). A similar clinical condition has not been recorded in dogs. Intestinal inflammation may escalate ascending bacterial infection from the duodenum into the liver and pancreas (Widdison, 1994). Notably, in cats, the single pancreatic duct and the bile duct join and enter the duodenum together, with increased chance of microbial infection leading to inflammatory disorders (Scherk, 2010). The duodenal resident bacterial load, mainly Escherichia coli (Simpson, 2012) is, therefore, markedly higher in the cat, compared to the dog (Rothrock, 2013). Vomiting may cause reflux entry of duodenal fluid into the pancreatic and bile ducts leading to cascading inflammatory bioepisodes (Twedt, 2014). In the pathogenesis of triaditis, an immune component is also stated to be involved (Clark et al., 2011).

Feline triaditis may be manifested in the acute or chronic form on presentation (Scherk, 2010). Physical examination reveals non-specific clinical signs, namely weight loss, anorexia, vomiting, lethargy, diarrhea, painful abdominal distension, icterus, palpable mass, and hepatomegaly (Marks, 2013). There is no breed/ gender predilection. Middle-aged mature cats are more susceptible. The diagnostic protocol includes cyanocobalamin (vitamin B_{12}) assay, hemogram, serum biochemistry panel, abdominal radiography and ultrasonography (Baez *et al.*, 1999; Dossin, 2011). The remedial strategy is focused on the major disease component. Supportive protocol includes oral rehydration fluid therapy, nutraceuticals back-up, adequate pain relief with amelioration of nausea and vomiting. Prognosis depends on the severity of disease, and improves significantly with timely diagnosis and treatment.

Case History and Observations

Trapper Sellers, 8 years old, slightly overweight (9.5 kg), domestic short hair neutered male cat was presented to the Milford Veterinary Clinic on September 1, 2020 for periodic health checks and vaccination schedule (feline leukemia, feline distemper and rabies) with no apparent health issues. Moderate dental tartar was noted. Physical examination revealed rectal temperature 100.8^o F, heart rate 160/minute, respiration rate 44/ minute, capillary refill time (CRT) <2 seconds and body condition sore (BCS) 4.5/5. Since, the patient was gradually gaining weight, a blood panel was recommended to the owner. The hemogram (Table 1) was normal, except marginally low neutropenia.

Diagnosis and Treatment

The abnormal values of the liver and gall bladder (Table 2) prompted in-clinic ultrasound probe into the abdominal internal organs (Fig.1-4) by the visiting imaging specialists (23.09.2020).

Veterinary Information Network (VIN) triaditis regimen (Rothrock, 2013) was followed. Nutraceuticals, Ursodiol generic 300 mg caps (AMEX LCI) and SAMe (S-adenosyl methionine) 200 mg caps, one each OD, PO continuously for 5 weeks promoted the natural cell

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Fig. 1. Echo profile of the patient's gall bladder.



Fig. 2. Echo profile of the patient's live



Fig. 3. Echo profile of the patient's intestine



Fig. 4. Echo profile of the patient's pancreas



Fig. 5. Common bile duct join the pancreatic duct and open into the duodenum (Jergens and Allenspach, 2016).

Table 2: Patient's blood chemistry panel

Parameter (Units)	Result	Range	Status	Parameter (Units)	Result	Range	Status
TEC (1x10 ⁶ / μl)	10.58	6.54-12.20	Normal	Glucose (mg/dL)	150	71-159	Normal
Hematocrit (%)	51.9	30.3	Normal	SDMA (µg/dL)	9	0-14	Normal
Hemoglobin (g/dL)	16.1	9.8-16.2	Normal	Creatinine mg/dL)	1.7	0.8-2.4	Normal
MCV (fL)	49.1	35.8-53.1	Normal	BUN (mg/dL)	23	16-36	Normal
MCH (g/dL)	15.2	11.8-17.3	Normal	BUN/Creatinine ratio	14		
MCHC (g/dL)	31.0	26.1-35.8	Normal	Phosphate (mg/dL)	4 5	3 1-7 5	Normal
Reticulocyte (%)	9.5			Calcium (mg/dL)	0.0	7.8-11.3	Normal
Reticulocyte $(1x10^{3/2})$	49.7	3.0-50.0	Normal	Total protein (g/dL)	10.5	5.7-8.9	High
μL) TLC (1-103/L)	5 20	2 97 17 02	N	Albumin (g/dI)	4.6	2 3-3 9	High
$ILC (1X107 \mu L)$	5.20 42.7	2.8/-1/.02	Normai	Glabulin (g/dL)	5.0	2.5 5.7	Uich
Neutrophil (%)	42.7			Globulin (g/dL)	5.9	2.8-3.1	підп
Lymphocyte (%)	50.2			A/G ratio	0.8		
Eosinophil (%)	4.2			ALT (U/L)	313	12-130	High
Monocyte (%)	2.1			ALKP (U/L)	343	14-111	High
Basophil (%)	0.8			GGT (U/L)	106	0-4	High
Neutrophil $(1 \times 10^{3} / \mu I)$	2.22	2.30-10.29	Low	Total Bilirubin (mg/L)	4.9	0-0.9	High
$I \text{ ymphocyte } (1 \times 10^3 / \text{ s})$	2.61	0 92-6 88	Normal	Cholesterol (mg/dL)	192	65-225	Normal
μ L)	2.01	0.92-0.88	Normai	Amylase (U/L)	97.3	500-1500	Normal
Eosinophil	0.22	0.17-1.57	Normal	Lipase (U/L)	428	100-1400	Normal
$(1x10^{3}/\mu L)$				Na ⁺ (mmol/L)	161	150-165	Normal
Monocyte $(1 \times 10^3 / \text{ mJ})$	0.11	0.05-0.67	Normal	K^+ (mmol/L)	3.6	3.5-5.8	Normal
Basophil $(1 \times 10^3 / \mu L)$	0.04	0.01-0.26	Normal	Na ⁺ / K ⁺ ratio 45			
Thrombocytes $(1 \times 10^3 / \mu L)$	307.0	151-600	Normal	Cl ⁻ (mmol/L)	121	112-129	Normal
				Total T4 (µg/dL)	2.1	0.8-4.7	Normal
IDEXX Laboratories Procyte Dx Auto cell counter				IDEXX Laboratories Catalyst Dx Autoanalyzer			

Table 1: Patient's hemogram

healing process. Cobalamin supplement (a) 250 μ g SC q for 7 days proved highly beneficial. Supportive therapy comprised use of home food, effective pain management with alleviation of nausea/ vomiting. Recovery was evidenced by the progressive improvement in body condition with increased activity.

Discussion

In the instant case, abdominal internal organs echo-profiles (Fig.1-4) indicated concurrent cholangiohepatitis, chronic pancreatitis and inflammatory bowel disease (IBD), named "triadits". The prognosis is less favourable in acute pancreatitis (Brister, 2020). The mute question is why triaditis is frequently observed in the cat, but not in the dog? The feline pancreatic duct and the common bile ducts join and enter the duodenum together (Fig. 5) with the markedly increased chance of bacterial translocation and infection (Jergens and Allenspach, 2020). Abdominal ultrasound proved crucial in the chance discovery of triaditis in the feline patient, Trapper Sellers before the onset of clinical symptoms. The owner is currently aware that if the symptoms flare up, the above treatment is available to the patient *im promptu* for the much-needed relief.

References

- Baez, J.L., Hendrick, M.J., Walker, L.M. and Washabau, R.J. (1999). Radiographic, ultrasonographic and endoscopic findings in cats with inflammatory bowel disease of the stomach and small intestine: 33 cases (1990-1997). J. Am. Vet. Med. Assoc., 215: 349-54.
- Brister, Jacqueline (2020). Triaditis in cats, Veterinary Partner, VIN.
- Clark, J.E.C., Haddad, J.L., Morgan, M.J and Brown, D.C. 2011. Feline cholangitis: a necropsy study of 44 cats (1986-2008). *J. Feline Med. Surg.*, **13** (8): 570-76.
- Dossin, O. (2011). Laboratory tests for diagnosis of gastrointestinal and pancreatic diseases. *Topics in companion Anim. Med.*, 26: 86-97.
- Fragkou, E.C., Adamama-Moraitou, K.K., Poutahidis, T., Prassinos, N.N., Kritsepi-Konstantinou, M., Xenoulis, P.G., Steiner, J.M., Lidbury, J.A., Suchodolski, J.S. and Rallis, T.S. (2016). Prevalence and clinicopathological features of triaditis in a prospective case series of symptomatic and asymptomatic cats. J. Vet. Intern. Med., 30 (4): 1031-45.

- Jergens, A.E. and Allenspach, K. (2016). Feline inflammatory gastrointestinal disease. In: *August's Consultations in Feline Internal Medicine*, 7: 129-136, Ed. Susan E. Little, Elsevier, St. Louis, MO, USA.
- Marks, S.L. (2013). Feline triaditis: current concepts. In: Proc. World Small Animal Veterinary Association (WSAVA) World Congress, Auckland, New Zealand.
- Rothrock, K. (2013). Triaditis, VINCyclopedia of Diseases, Veterinary Information Network (VIN), USA. Revised 2020.
- Scherk, M. (2010). Update on feline gastrointestinal syndromes; pancreatitis and triaditis. In : *Proc. Western Veterinary Conference*, Las Vegas, NV, USA.
- Simpson, K.W. (2012). Is there a direct link between IBD, cholangitis, and pancreatitis in cats? In: *Proc. 22nd Congress of the European College of Veterinary Internal Medicine-Companion Animals*, Toulouse, France.
- Simpson, K.W. (2015). Pancreatitis and triaditis in cats: cause and treatment. J. Small Anim. Pract., 56 (1), 40-49.
- Twedt, D. (2012). Update on feline liver disease. In: *Proc. Atlantic Coast Veterinary Conference*, Atlantic City, NJ, USA.
- Widdison, A.L., Karanjia, N.D. and Reber, H.A. (1994). Routes of spread of pathogens into the pancreas in a feline model of acute pancreatitis. *Gut*, **35** (9): 1306-10.

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