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Suffering in silence: spotting signs of chronic feline triaditis

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Conflicts of Interest & Disclaimer

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Agenda

- + Aetiology of triaditis
- + Review of the literature
- + Diagnostic approach to case with suspected triaditis
- + Treatment plan

+ Case Studies





What is triaditis?

+ Defined as concurrent pancreatitis, cholangitis and chronic enteropathy

 Reported in approximately 17–39% of ill cats in referral populations





- + Poorly understood
- + ?infectious
- + ?autoimmune
- + ?duct obstruction
- + ? different diseases in each organ or if the disease process in each organ has the same aetiology





- + Poorly understood but increased risk of bacteria ascending duodenum
- + Feline cf canine
 - + Shorter GIT in cats
 - + Higher concentration bacteria in duodenum
 - + Entrance of pancreatic duct
- + Close association between the pancreas and the duodenum





- + Ascending infection from duodenum into liver is postulated
- + Systemic inflammatory response syndrome can result in translocation of enteric bacteria resulting in a neutrophilic cholangitis





- + In one study (Twedt et al 2013) bacteria were noted in 21/39 (54%) cats with inflammatory liver disease.
- + The predominant bacteria identified were common enteric forms, such as *E coli* and *Enterococcus* species.





- + ?Intestinal inflammation as the inciting cause
- + The most common type of intestinal inflammation is either lymphoplasmocytic inflammation or small cell lymphoma.
- + Intestinal disease may lead to increased intestinal permeability contributing to bacterial translocation







- + Obstruction to the flow of bile or pancreatic juices may also play a role in triaditis
- + Blockage or dysfunction of the sphincter can predispose cats to pancreatitis and cholangitis



Previous studies

- + Many studies PM based
- + 1996 study found that 39% of cats diagnosed with cholangitis also had IBD and pancreatitis (i.e., triaditis)
 Concurrent nephritis was found in 33% of cats with cholangitis
- + 2011 study of 44 cats with moderate to severe cholangitis found that inflammatory bowel disease (50%), pancreatitis (60%), or both (32%) commonly accompanied cholangitis. Concurrent nephritis was found in 81% of cats with cholangitis

Weiss DJ, Gagne JM, Armstrong PJ. Relationship between inflammatory hepatic disease and inflammatory bowel disease, pancreatitis, and nephritis in cats. J Am Vet Med Assoc. 1996 Sep 15;209(6):1114-6. PMID: 8800259. Callahan Clark JE, Haddad JL, Brown DC, Morgan MJ, Van Winkle TJ, Rondeau MP. Feline cholangitis: a necropsy study of 44 cats (1986-2008). J Feline Med Surg. 2011 Aug;13(8):570-6. doi: 10.1016/j.jfms.2011.05.002. Epub 2011 Jun 29. PMID: 21719332; PMCID: PMC10822413.



Prospective study

- + Biopsies performed on 27 symptomatic cats with chronic clinical signs that could be attributed to triaditis
- + Biopsies also performed in 20 asymptomatic cats presented for ovariohysterectomy.

Fragkou FC, Adamama-Moraitou KK, Poutahidis T, Prassinos NN, Kritsepi-KonsPrevalence and Clinicopathological Features of Triaditis in a Prospective Case Series of Symptomatic and Asymptomatic Cats. J Vet Intern Med. 2016 Jul;30(4):1031-45. doi: 10.1111/jvim.14356. Epub 2016 Jun 14. PMID: 27296565; PMCID: PMC5089651tantinou M, Xenoulis PG, Steiner JM, Lidbury JA, Suchodolski JS, Rallis TS..

Prospective study

+ Symptomatic cats

+ Histopathological evidence of inflammation in 1, 2, or all 3 organs was present in 27/39 (69.2%)

+ Asymptomatic cats

- + Histopathological lesions of either cholangitis or enteritis alone, or combinations of inflammatory infiltration in 2 of the 3 organs were identified in the absence of clinical signs in 20/39 (51.3%).
- + Pancreatitis alone or triaditis was only present in cats that were symptomatic.

Fragkou FC, Adamama-Moraitou KK, Poutahidis T, Prassinos NN, Kritsepi-KonsPrevalence and Clinicopathological Features of Triaditis in a Prospective Case Series of Symptomatic and Asymptomatic Cats. J Vet Intern Med. 2016 Jul;30(4):1031-45. doi: 10.1111/jvim.14356. Epub 2016 Jun 14. PMID: 27296565; PMCID: PMC5089651tantinou M, Xenoulis PG, Steiner JM, Lidbury JA, Suchodolski JS, Rallis TS..



Clinical Presentation

- + Vague clinical signs including weight loss, lethargy and anorexia.
- + Anorexia 63-97% cases
- + Lethargy 28-100% cases
- + More specific signs such as icterus and vomiting may be noted.
- + Pyrexia, abdominal pain, hepatomegaly possible
 - + 25% pancreatitis cases pyrexic and 50% hypothermic





Consistent signs



- + Full haematology, serum biochemistry
- + SNAP fPLi, Quantitative fPLi or DGGR lipase measurement
- + Folate/B12 assessment
- + Abdominal imaging
 - + Radiographs have low sensitivity
 - + Ultrasound is superior
- + For a definitive diagnosis, biopsies are required



Blood work



- + Non specific changes on routine blood work.
- + May see non regenerative anaemia
- + Liver parameters can be normal or increased
- + Ionised calcium should be assessed in cats with acute pancreatitis



Pancreatic tests

+ In house

- + Lipase
- + Amylase
- + fPLi- SNAP
- + Catalyst Pancreatic lipase

+ At lab

- + Quantitative Spec[®] fPL[™]
- + DGGR lipase
- + fTLI



- + fPLi SNAP good to rule out
- + fTLI, lipase, amylase not useful to confirm pancreatitis



Catalyst[®] Pancreatic Lipase Test

- + Activity assay cf Spec[®] fPL[™] which is an immunoassay
- + Catalyst[®] Pancreatic Lipase Test aligns well with Spec[®] cPL[™] and Spec[®] fPL[™] tests
- + One test validated for both dogs and cats
- + Real-time diagnoses with results in less than 10 minutes





Diagnostic imaging

- + Ultrasound is superior to radiographs
- + In pancreatitis often see thick left limb of the pancreas, severely irregular pancreatic margins, and hyperechoic peripancreatic fat
- With cholangitis may see heterogenous liver parenchyma +/- biliary duct obstruction
 + Normal liver does not rule out cholangitis
- + Inflammatory bowel disease may result in loss of intestinal layering, thickening of the muscularis layer, generalised intestinal thickening and mesenteric lymphadenopathy



Treatment

- + Depends on severity of signs
- + Histopathology and culture can guide treatment
- + Supportive treatment often required
 - + Fluid therapy
 - + Analgesia
 - + Anti emetics
 - + Feeding tube placement





Treatment for pancreatitis

- + Analgesia
- + Anti-emetics
- + Lack of evidence regarding low fat diets
- + Consider placement of feeding tube
 - + NO vs O tube
 - + Start at 1/3 RER divided into several meals





Treatment for cholangitis/cholangiohepatitis

- + Depends on the histopathology and culture
- + Most common types are lymphocytic cholangitis or neutrophilic cholangitis.
- + Supportive care similar to treatment of pancreatitis
- + Additional therapy considered
- + Vitamin K1
- + Anti oxidants such as SAMe





Neutrophilic Cholangitis

- + Occurs in younger cats
- + Antibiotic therapy ideally based on culture and sensitivity
- + Culture however can be falsely negative
- +?FISH





Lymphocytic Cholangitis

- + Chronic and slowly progressive disease
- + FISH studies have failed to find underlying bacterial aetiology
- + Thought to be underlying immune mediated aetiology
- + Prednisolone mainstay of therapy
 - + Start at 1-2mg/kg/day and taper after 4-6weeks





Inflammatory bowel disease

- + Thought to be due to hypersensitivity reactions to dietary or bacterial antigens.
- + Diet trial initially
 - + May need multiple diets
- + Immunomodulatory therapy is mainstay of treatment
- + Supplementation of cobalamin if indicated





Max

- + 10 year old MN Siamese
- + Outdoor access
- + UTD vaccinations and flea/worming treatment
- + 1 month history of intermittent vomiting and lethargy
- + 1 week history of anorexia
- + No response to short courses of antibiotic (synulox) and Metacam.





Haematology/Serum biochemistry

- + Neutrophilia
 - + Infection, inflammation
- + Anaemia
 - + Chronic disease deemed most likely
- + Raised ALT, ALP, bile acids
 - + Primary hepatic disease or secondary hepatopathy due to GI disease
- + Hypoalbuminaemia
 - + Negative acute phase protein
 - + Lost via GIT, renal or reduced production in liver





Investigations

- + Suspect hepatic and/or GI disease
- + Cannot rule out secondary hepatopathy
- + Cannot rule out concurrent pancreatitis
- + Further investigations with Spec[®] fPL[™], folate and cobalamin performed
- + Urinalysis performed to rule out proteinuria as a cause of the hypoalbuminaemia





Results

- + Spec[®] fPL[™] raised
 - + Primary pancreatitis or secondary to GI/liver disease
- + Cobalamin reduced
- + Folate within normal limits
- + Urinalysis unremarkable; UPC 0.2





Advanced imaging

- + Abdominal ultrasound
- + Hypoechoic enlarged pancreas with hyperechoic mesenteric and peripancreatic fat
- + Normal appearance of liver and biliary tree
- + Thickening of muscularis layer in small intestine, predominately in the ileum and jejunum





Options

+ Trial treatment?

- + Endoscopy and ultrasound guided FNA of liver and bile aspirate?
- + Exploratory laparotomy?





Exploratory laparotomy

- + Coagulation profile performed first and was wnl
- + Multiple biopsies taken
- + Biopsies submitted for histopathology
- + Bile aspirate submitted for culture and sensitivity and cytology
- + Placement of oesophageal feeding tube





Post op treatment

+ IVFT

- + Analgesia buprenorphine
- + Anti emetic- maropitant
- + Enteral feeding
- + Potentiated amoxicillin IV pending results
- + B12 supplementation
- + SAMe and UDCA started





Results

- + Lymphoplasmocytic gastritis and enteritis
- + Moderate chronic pancreatitis
- + Neutrophilic cholangiohepatitis
- + *E.Coli* cultured from bile
 - + Sensitive to all antibiotics tested against





Treatment

- + Continue enteral feeding until appetite returns
- + Once appetite back to normal transition to hydrolysed diet e.g. Purina HA
- + Continue potentiated amoxicillin for 6-8 weeks
- + Continue SAMe and UDCA





Follow up

- + O tube removed after 3 weeks
- + Exclusively fed Purina HA
- + Resolution of vomiting and weight gain at revisit 8 weeks later
- + Continued solely on dietary therapy



Follow up

- + 6 months later represented for vomiting and small bowel diarrhea
- + Continued to be fed on Purina HA
- + Full blood work, faecal analysis and abdominal ultrasound performed.





Results

- + Blood work unremarkable
- + Folate wnl, cobalamin reduced again
- + Spec[®] fPL[™] increased
- + Faecal analysis unremarkable
- + Abdominal ultrasound revealed normal hepatic and biliary tree, thickening of intestinal muscularis and normal pancreatic parenchyma.
- + FNA of liver and bile aspirate performed.
 - + Cytology unremarkable and culture negative





Diagnosis

- + Chronic enteropathy
- + Possible pancreatitis
- + No evidence for return of bacterial cholangiohepatitis





Treatment/follow up

- + Purina HA
- + Prednisolone started at 2mg/kg/day
- + Vomiting and diarrhea improved over a month
- + Prednisolone tapered by 25% every 4 weeks and stopped
- + Long term dietary therapy



Alonzo – 9y MN DLH

- + 1 month history of weight loss (1Kg)
- + Otherwise no concerns
- + UTD vaccinations and flea treatment
- + No travel history
- + Indoor/outdoor access.
- + Not known to hunt





Physical examination

- + Bright, alert and responsive
- + Visibly icteric skin, mucous membranes

+ TPR wnl

+ Thoracic and abdominal palpation unremarkable

+ LN wnl







Haematology

	*RBC	9.33	10^12/L	7.12 - 11.46
	*Haematocrit	0.415	I/L	0.282 - 0.527
	*Haemoglobin	12.5	g/dL	10.3 - 16.2
	*MCV	44.5	fL	39.0 - 56.0
	*MCH	13.4	pg	12.6 - 16.5
	*MCHC	30.1	g/dL	28.5 - 37.8
	*Reticulocytes	8.4	10^9/L	<=50.0
Low	*Reticulocyte Haemoglobin	14.1	pg	15.3 - 22.9
	*WBC	11.8	10^9/L	3.9 - 19.0
	*% Neutrophils	71.0	%	
	*% Bands	2.0	%	
	*% Lymphocytes	17.0	%	
	*% Monocytes	9.0	%	
	*% Eosinophils	1.0	%	
	*% Basophils	0.0	%	
	*Neutrophils	8.38	10^9/L	2.62 - 15.17
	*Bands	0.24	10^9/L	0.00 - 0.30
	*Lymphocytes	2.01	10^9/L	0.85 - 5.85
High	*Monocytes	1.06	10^9/L	0.04 - 0.53
	*Eosinophils	0.12	10^9/L	0.09 - 2.18
	*Basophils	0.00	10^9/L	0.00 - 0.10
	*Platelets	302	10^9/L	155 - 641



Biochemistry

	Glucose	5.1	mmol/L	3.9 - 8.0	
	IDEXX SDMA	8	ug/dL	1 - 14	
	Creatinine	130.0	umol/L	80.0 - 203.0	
High	Urea	11.4	mmol/L	2.5 - 9.9	
	Phosphorus	1.51	mmol/L	0.90 - 2.20	
	Calcium	2.70	mmol/L	2.05 - 2.95	
	Magnesium	0.90	mmol/L	0.80 - 1.00	
	Sodium	155.0	mmol/L	145.0 - 157.0	
	Potassium	4.55	mmol/L	3.50 - 5.50	
	Na: K Ratio	34.07		28.00 - 40.00	
	Chloride	119.7	mmol/L	100.0 - 124.0	
High	Total Protein	83.4	g/L	60.0 - 80.0	
	Albumin	34.4	g/L	25.0 - 45.0	
High	Globulin	49.0	g/L	25.0 - 45.0	
	Albumin: Globulin Ratio	0.70		0.60 - 1.50	
High	ALT	441.8	U/L	5.0 - 60.0	
High	AST	207.0	U/L	10.0 - 50.0	
High	ALP	145.0	U/L	<=60.0	
High	GGT	5.3	U/L	2.0 - 5.0	
High	Bilirubin - Total	81.0	umol/L	<=5.1	
High	Cholesterol	7.10	mmol/L	2.20 - 4.00	



Abdominal ultrasound













Further investigations

- + Coagulation profile
 - + PT 14.2 seconds (9-14)
 - + APTT 44 seconds (28-44)
- + Liver FNA
- + Bile aspirate
- + Bile cytology and culture
- + No significant abnormalities
 - + ?false negative result





Screening for concurrent diseases



- + Folate wnl
- + Cobalamin 255ng/L (ref >400)
- + Spec[®] fPL[™] 9 (ref <4)
- + Suggestive of possible triaditis



Next step

+ Trial treatment

- + Treatment for suspected enteropathy
- + Treatment for liver disease ?inflammatory ?infectious ?other
- + Pursue definitive diagnosis
 - + Exploratory laparotomy with biopsies





Exploratory laparotomy





Images courtesy of Professor Donald Yool



Histopathology results

- + Lymphocytic cholangitis
- + Stomach nad
- + Duodenum, ileum and jejunum lymphoplasmacytic enteritis
- + Bile and liver culture negative
- + Pancreas inflamed at time of surgery. Biopsy not taken

+ Results suggestive of triaditis



Treatment

- + B12 supplementation
- + SAMe

+

- + Hydrolysed diet
- + Prednisolone therapy





Outcome

- + Prolonged recovery post surgery
- + Once steroids started responded well to treatment
- + Transitioned to chlorambucil due to concern regarding DM
- + Long term dietary management and close monitoring



Summary

- + Controversy still exists over the underlying aetiology and initial initiating cause
- + Clinical signs can be vague
- + Screen for concurrent diseases, particularly chronic enteropathy

- + Treatment should ideally be guided by histopathology and culture results
- + Cats often require intensive hospitalization and supportive care





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