Veterinary Record casereports

Exocrine pancreatic insufficiency with concurrent pancreatitis, inflammatory bowel disease and cholangiohepatitis in a cat

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Complete List of Authors:	Costa Devoti, Chiara; Small Animal Teaching Hospital University of Liverpool-Leahurst Campus, Murtagh, Kevin; Small Animal Teaching Hospital, University of Liverpool, Internal medicine Batchelor, Daniel; Small Animal Teaching Hospital, University of Liverpool, Internal medicine; Small Animal Teaching Hospital, University of Liverpol, Internal medicine Silvestrini, Paolo; Small Animal Teaching Hospital, University of Liverpool, Internal medicine
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Abstract:	A 5-year-old male Persian cat was referred for chronic weight loss, polyphagia and clay-coloured abundant stools. Biochemistry showed elevated liver enzymes activity, hypocholesterolaemia and hypocobalaminaemia. On abdominal ultrasound, thickened small intestine wall and tortuous biliary tract were detected. Pancreas was difficult to visualise. Surgical biopsies of small intestine, liver and pancreas and low fTLI allowed a final diagnosis of pancreatitis, inflammatory bowel disease, cholangiohepatitis and exocrine pancreatic insufficiency. The cat was treated with antibiotics, prednisolone, oral pancreatic enzymes and parenteral cobalamine. At one-year follow-up, the medical treatment has led to a complete resolution of clinical signs.

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SUMMARY

A 5-year-old male Persian cat was referred for chronic weight loss, polyphagia and clay-coloured abundant stools. Biochemistry showed elevated liver enzymes activity, hypocholesterolaemia and hypocobalaminaemia. On abdominal ultrasound, thickened small intestine wall and tortuous biliary tract were detected. Pancreas was difficult to visualise. Surgical biopsies of small intestine, liver and pancreas and low fTLI allowed a final diagnosis of pancreatitis, inflammatory bowel disease, cholangiohepatitis and exocrine pancreatic insufficiency. The cat was treated with antibiotics, prednisolone, oral pancreatic enzymes and parenteral cobalamine. At one-year follow-up, the medical treatment has led to a complete resolution of clinical signs.

BACKGROUND

Exocrine pancreatic insufficiency (EPI) is a condition caused by reduced or absent synthesis and secretion of pancreatic enzymes, leading to signs of maldigestion. Different pancreatic diseases can cause EPI, such as chronic pancreatitis, pancreatic acinar atrophy, pancreatic neoplasia and obstructions of the pancreatic duct (Wibarg, 2013). Moreover in Greyhound dogs an uncommon form of juvenile pancreatic atrophy causing both EPI and insulin dependent diabetes mellitus has been recently described (Brenner, 2009). EPI has been rarely reported in cats (Steiner, 2012; Wibarg, 2013) and chronic pancreatitis is considered the most common cause in this species (Steiner, 2012; Thompson et. al., 2009). It may become a more common diagnosis nowadays that a validated pancreatic function test is widely available. Moreover in the feline species is common the association of pancreatitis, inflammatory bowel disease (IBD) and cholangitis/cholangio-hepatitis, sometimes called triaditis (Cattin, 2013; Elwood, 2010; Weiss et. al., 1996). The present report describes the successful management of a cat diagnosed with these three pathologies and concurrent EPI.

CASE PRESENTATION

A 5-year-old male neutered Persian cat was presented for a 2 months history of weight loss, polyphagia, lethargy and soft and clay-coloured abundant stools. On physical examination the cat was quiet but reactive. His body weight was 2.19 kg with a decreased body condition score (2/9). His hair coat was greasy and wet looking especially in the hind limbs, perianal

and tail regions (Figure 1). Thoracic auscultation was unremarkable. The abdomen appeared enlarged and pendulous. Mildly thickened small intestinal loops and mild discomfort were detected during cranial abdominal palpation. No thyroid nodules were palpated in the neck.

INVESTIGATIONS

Haematology and urine analysis were unremarkable. Biochemistry profile showed increased ALT (58 IU/l; RI:7-50) and ALP (41 IU/l; RI: 0-40), decreased cholesterol (1.8 mmol/l; RI 1.9-2.9), decreased total protein (53 g/l; RI:55-78) and serum cobalamine (83.1 pg/ml; RI:150-350). Free and total T4 were within reference intervals. During hospitalisation, the cat passed voluminous, malodorous and yellow-coloured stools (Figure 2). The latter alongside the ravenous appetite (Video 1) raised the suspicion of EPI that was confirmed by decreased fTLI value (2.6 ug/L; RI: 12.1-82.0 ug/L).

Thoracic (Figure 3) and abdominal (Figure 4) radiographs showed respectively enlarged sternal lymph nodes and diffuse loss of serosal detail, consistent with peritoneal effusion and poor body condition. Abdominal ultrasound showed a thickened gallbladder wall and tortuous intra and extra hepatic biliary ways (Figure 5), increased thickness of the small intestinal muscularis and hypoechoic mucosa (Figure 6), mesenteric and colic lymphadenopathy and confirmed a small volume of free peritoneal fluid (Figure 7).

In order to further investigate the abdominal abnormalities, an exploratory laparotomy with full thickness intestinal, hepatic, gallbladder and pancreatic biopsies was performed.

Histological evaluation of the liver and gall bladder revealed a lymphocytic-neutrophilic colangiohepatitis and cholecystitis. Smears of gall bladder aspirates showed numerous bacterial rods, frequently in chains and the cytological findings were compatible with cholecystitis. Heavy growth of haemolytic *Escherichia coli* was obtained from the bile culture and a susceptibility test was performed. Small intestinal and pancreatic histopathology was consistent with lymphoplasmacytic to neutrophilic enteritis and severe, chronic pancreatitis, respectively. In addition, there was a marked, diffuse loss of pancreatic acinar cells with replacement by areas of fibrosis compatible with a diagnosis of pancreatic atrophy.

DIFFERENTIAL DIAGNOSIS

Based on presentation, biochemistry and imaging findings the differential diagnosis were numerous.

Diarrhoea may be caused by enteritis caused by food intolerance, bacterial (*Salmonella*, *Clostridium*, *E. coli* and *Campilobacter* species) or parasitic infection (Helmints and Protozoans), viral infection (Fiv, Felv or FIP), idiopathic IBD, intestinal neoplasia (mainly lymphoma, carcinoma and adenocarcinoma), intestinal obstruction or dysmotility, liver and kidney diseases, hypoadrenocorticism, hyperthyroidism, pancreatitis, pancreatic acinar atrophy, obstructions of the pancreatic duct and EPI. Yellow-coloured, voluminous stools are quite typical of EPI. Polyphagia can be found in cats with hyperthyroidism, diabetes mellitus, intestinal malabsorption and EPI. Weight loss may be caused by hyperthyroidism, neoplasia, inadequate caloric ingestion, heart disease, CKD, enteropathies, hepatic and pancreatic

diseases.

Elevation of liver enzyme activities can be found in cats with acute or chronic inflammatory liver disease (cholangitis and/or cholangio-hepatitis), hepatic infection or toxicity, hepatic lipidosis and obstruction of bile duct. Hypoalbuminemia may be caused by decreased proteins production or absorption, decreased food intake, increased intestinal or renal loss.

Hypocholesterolaemia primarily occurs with protein-losing enteropathies, hepatic insufficiency, neoplasia and severe malnutrition. Low serum cobalamine can be caused by distal or diffuse small intestinal disease involving the ileum, small intestinal dysbiosis, overgrowth, EPI, hereditary cobalamine malabsorption or low dietary intake. Decreased fTLI value are indicative of EPI.

The histological evaluation confirmed the combination of enteritis, pancreatitis and cholangitis. The loss of exocrine pancreatic tissue possibly secondary to chronic pancreatitis was the likely cause of EPI.

TREATMENT

The cat was treated with amoxicillin/clavulanic acid (25 mg/kg BID) and prednisolone (2 mg/kg SID) during 6 weeks and metronidazole (12.5 mg/kg BID) during 10 days. This association of steroids an antibiotics was justified by a diagnosis of a mixed form of neutrophilic to lymphocytic cholangiohepatitis. The choice of amoxicillin/clavulanic acid as antibiotic was based on bile culture results. Pancreatic enzymes were mixed twice daily with high digestible, low fat diet and cobalamine (250 mcg) was given sub-coutaneously once weekly for a total of six weeks and then monthly life-long. The cat was discharged after 5 days of hospitalisation with an improved body weight (2.4 kg).

OUTCOME AND FOLLOW-UP

At the time of the first re-check, 10 days after the discharge, the cat was still passing yellow-soft faeces but was more active and had gained weight (2.9 kg). Therapy was continued as before. At 1-month recheck the BCS was improved (3/9). Biochemistry profile was unremarkable but folate concentration was increased (73 ng/ml, RI: 9-24 ng/ml) suggesting a possible small intestinal dysbiosis. Metronidazole was then re-started. Over the next 5 months the cat became gradually less polyphagic and faecal consistency and colour normalised. The hair-coat improved as well. Prednisolone was progressively tapered over 2 months then stopped. The metronidazole was also stopped after 2 months. Pancreatic enzymes and cobalamine supplementation was continued. After one year the cat is bright, passing normal faeces, with a BCS of 4/9 and his body weight is 3.8 kg.

DISCUSSION

EPI is a disease rarely reported in cats and chronic pancreatitis is considered to be the most common cause (Wiberg, 2013; Steiner, 2012). Pancreatic enzymes play an important role in assimilation of food and their absence causes maldigestion. Large molecules of undigested nutrients remaining in the small intestinal cause an osmotic pull of water exceeding the absorption. This leads to steatorrhea, loose, voluminous and malodorous faeces with a

yellow- to clay-coloured appearance (Thompson et al., 2009; Steiner, 2012). As in this case, the high fat content of the stools can lead to a greasy appearance of the hair coat particularly in the perianal and tail region (Steiner, 2012). Inadequate digestion and assimilation of nutrients cause a negative energy balance leading to weight loss and a compensatory polyphagia (Thompson et al., 2009).

Clinical diagnosis is confirmed with validated pancreatic function test. Serum fTLI is a highly species-specific test that quantifies the amount of trypsinogen and related molecules concentration in the plasma. Values <8 µg/L (RI: 12.0 to 82.0 µg/L) are considered diagnostic for EPI in cats (Steiner, 2012). Now that a validated test for cats is easily available the diagnosis of EPI may become more common.
Other common biochemistry abnormalities are hypocholesterolaemia, hypocobalaminaemia and elevated folate concentration. Less common abnormalities associated to EPI are hyperglycaemia, in case of concurrent diabetes mellitus (Larsen, 1993), hyperammonaemia (Watanabe et al., 2012), hypokalaemia (Daste et al., 2014) and vitamin K – responsive coagulopathy (Perry et. al., 1991).

Cobalamine deficiency in cats with EPI is likely caused by insufficient pancreatic production of intrinsic factor that is necessary for ileal absorption of cobalamine. The intrinsic factor is only produced by the pancreas in the feline species. Moreover, pancreatic enzymes deficiency can also lead to failure in liberating vitamin B12 from binding R-protein in the duodenum (Thompson et al., 2009).

Increased folate number in this animal may have multiple causes. Loss of pancreatic enzymes creates a small intestinal environment optimal for secondary bacterial overgrowth and intestinal dysbiosis due to presence of undigested molecules. An abnormal high number of intestinal bacteria synthesizes and releases folate (Westermarck et. al., 2005). Moreover hypocobalaminaemia may cause decreased folate utilization because cobalamine is needed for conversion of methylfolate to the active form tetrahydrofolate, which is required for DNA synthesis. In addition, an abnormally high numbers of bacteria in the duodenum results in competition for cobalamine, creating a vicious circle (Thompson et al., 2009).

Intestinal dysbiosis, also called bacterial overgrowth (SIBO), is described as an increase in the absolute number of bacteria in the upper SI during the fasting state. Dysbiosis in humans arises secondary to a number of underlying disorders that interfere with intestinal motility, substrate availability and bacteriostatic/cidal secretions. The exactly diagnostic criteria for dysbiosis in companion animals are still debated, nonetheless a genuine bacterial overgrowth exist in conditions equivalent to those in humans (German, 2013). Intestinal dysbiosis may be idiopathic primary (antibiotic responsive diarrhoea) or secondary to an underlying pathology, such as EPI (Thompson et al., 2009). Increased duodenal bacteria have already been reported in cats with EPI (Perry et. al., 1991) and secondary dysbiosis

was likely the cause of the increase folate level in this cat.

Although during secondary dysbiosis appropriate treatment for the underlying pathology is preferable, in some clinical cases concurrent antibacterial therapy is necessary. The choice of the most appropriate antibiotic is controversial, however tylosin and metronidazole are commonly used thanks their spectrum of activity and immunomodulatory activities. Prolonged therapy is often necessary (German, 2013). This cat has been treated with 8 weeks of metronidazole after a first relapse and the prolonged therapy associated to enzyme supplementation led to a resolution of clinical signs.

The association between pancreatitis, IBD and inflammatory hepatic diseases is a common finding in cats (Weiss et. al., 1996) and it is sometimes referred as triaditis, but this terminology is still controversial (Cattin, 2013; Elwood, 2010). The specific conditions of each organ that constitute a diagnosis of triaditis remains to be defined. In fact in actual scientific literature cat triaditis range from any inflammatory process within these organs, to the combination of chronic pancreatitis, chronic cholangitis/cholangiohepatitis and IBD. In any case, the definitive diagnosis of "triaditis" is based on the histopathological evaluation of each organ (Simpson, 2015).

IBD refers to persistent gastro-intestinal signs and histopathologic evidence of chronic intestinal inflammation, causing malabsorption and maldigestion. It is only appropriate to use the term idiopathic IBD if no underlying cause for the inflammation can be found. (German, 2013). Its precise aetiology is still controversial but IBD may be caused by an inappropriate immune response to dietary and/or bacterial antigens presented to the gastrointestinal mucosa (Elwood, 2010). IBD can be further classified based on the predominant inflammatory cells type at histological examination of intestinal biopsies. In this case the patient had a lymphoplasmacytic to neutrophilic enteritis, a quite common histological diagnosis in cats (German, 2013).

In the 80% of cats the major pancreatic and common bile duct form a unique final duct exiting into the duodenum with a common papilla. This particular anatomical feature can allow a reflux of duodenal fluid containing bacteria, bile salts, and activated pancreatic enzymes in both the pancreatic and biliary system, causing inflammation and/or infection (Washabau, 2013). It has also been suggested that an haematogenous spread can be an additional cause of feline inflammatory liver disease (Twedt et al., 2014).

In this case the histology showed a lymphocytic-neutrophilic colangiohepatitis and cholecystitis. Bile culture was also positive for haemolytic *Escherichia coli.* Feline hepatobiliary inflammatory diseases, principally cholangitis, have been generally classified as neutrophilic (acute and chronic), lymphocytic and associated with liver flukes (Twedt et al., 2014), the neutrophilic form being the one most commonly diagnosed at histological examination (Callahan et al., 2011) . In the present case a mixed form of neutrophilic and lymphocytic cholangiohepatitis was diagnosed and for this reason the patient received a

combined therapy with antibiotics and steroids.

The exact aetiology of pancreatitis in cats is still not clear and there are a number of potential triggers of pancreas inflammation (enteric bacteria, immune-mediated diseases, idiosyncratic drug reactions, diet, idiopathic causes and others). It is not even precisely known if pancreatitis is caused by enteritis/colangiohepatitis or if it may be the initiating stimulus for triaditis (Simpson, 2015). Histopathologic criteria for the classification of pancreatitis have not been universally standardized in veterinary medicine and different histopathologic scoring systems have been proposed for both cats and dogs (De Cock et al, 2007; Hess et al, 1998). Acute pancreatitis ranges from oedematous to necrotising and it can progress to chronic pancreatitis characterized by fibrosis, inflammation and atrophy (De Cock et al, 2007), and then to exocrine pancreatic insufficiency (Simpson, 2015).The histopathology of the pancreatic samples of this cat showed severe chronic pancreatitis and a diffuse loss of acinar cells replaced by areas of fibrosis. It is then likely that chronic pancreatitis caused a progressive loss of exocrine pancreatic cells, leading to a lack of pancreatic enzymes and development of typical clinical signs of EPI.

LEARNING POINTS/TAKE HOME

- EPI is rare in cats and the most common cause is chronic pancreatitis
- Typical clinical signs in cats with EPI are weight loss, polyphagia, clay-coloured abundant stools and greasy-looking coat on the hind limbs, perianal and tail regions.
- Therapy for concurrent pancreatitis, inflammatory bowel disease and cholangiohepatitis cats may include a combination of antibiotic, immune-suppressive drugs and diet change. Therapy for EPI consists in life-long supplementation of pancreatic enzymes.
- Cats with EPI have frequently low level of vitamin B12, therefore cobalamine should be supplemented by monthly sub-cutaneous injection.
- Intestinal dysbiosis is a common consequence of EPI and appropriate prolonged antibiotic therapy is often required.

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FIGURE/VIDEO CAPTIONS

Figure 1. Greasy and wet looking of the coat
Figure 2. Clay-coloured appearance of the stools
Figure 3. Right – lateral X-ray of the thorax with enlarged sternal lymph nodes
Figure 4. Right – lateral X-ray of the abdomen, diffuse loss of serosal detail
Figure 5. Abdominal ultrasound: thickened gallbladder wall and tortuous intra and extra hepatic biliary ways
Figure 6. Abdominal ultrasound: increased thickness of the small intestinal muscularis and hypoechoic mucosa
Figure 7. Abdominal ultrasound: mesenteric lymphadenopathy and free peritoneal fluid
Video 1. Polyphagia

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SUMMARY

A 5-year-old male Persian cat was referred for chronic weight loss, polyphagia and clay-coloured abundant stools. Biochemistry showed elevated liver enzymes activity, hypocholesterolaemia and hypocobalaminaemia. On abdominal ultrasound, thickened small intestine wall and tortuous biliary tract were detected. Pancreas was difficult to visualise. Surgical biopsies of small intestine, liver and pancreas and low fTLI allowed a final diagnosis of pancreatitis, inflammatory bowel disease, cholangiohepatitis and exocrine pancreatic insufficiency. The cat was treated with antibiotics, prednisolone, oral pancreatic enzymes and parenteral cobalamine. At one-year follow-up, the medical treatment has led to a complete resolution of clinical signs.

BACKGROUND

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and tail regions (Figure 1). Thoracic auscultation was unremarkable. The abdomen appeared enlarged and pendulous. Mildly thickened small intestinal loops and mild discomfort were detected during cranial abdominal palpation. No thyroid nodules were palpated in the neck.

INVESTIGATIONS

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TREATMENT

The cat was treated with amoxicillin/clavulanic acid (25 mg/kg BID) and prednisolone (2 mg/kg SID) during 6 weeks and metronidazole (12.5 mg/kg BID) during 10 days. This association of steroids an antibiotics was justified by a diagnosis of a mixed form of neutrophilic to lymphocytic cholangiohepatitis. The choice of amoxicillin/clavulanic acid as antibiotic was based on bile culture results. Pancreatic enzymes were mixed twice daily with high digestible, low fat diet and cobalamine (250 mcg) was given sub-coutaneously once weekly for a total of six weeks and then monthly life-long. The cat was discharged after 5 days of hospitalisation with an improved body weight (2.4 kg).

OUTCOME AND FOLLOW-UP

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DISCUSSION

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Other common biochemistry abnormalities are hypocholesterolaemia, hypocobalaminaemia and elevated folate concentration. Less common abnormalities associated to EPI are hyperglycaemia, in case of concurrent diabetes mellitus (Larsen, 1993), hyperammonaemia (Watanabe et al., 2012), hypokalaemia (Daste et al., 2014) and vitamin K – responsive coagulopathy (Perry et. al., 1991).

Cobalamine deficiency in cats with EPI is likely caused by insufficient pancreatic production of intrinsic factor that is necessary for ileal absorption of cobalamine. The intrinsic factor is only produced by the pancreas in the feline species. Moreover, pancreatic enzymes deficiency can also lead to failure in liberating vitamin B12 from binding R-protein in the duodenum (Thompson et al., 2009).

Increased folate number in this animal may have multiple causes. Loss of pancreatic enzymes creates a small intestinal environment optimal for secondary bacterial overgrowth and intestinal dysbiosis due to presence of undigested molecules. An abnormal high number of intestinal bacteria synthesizes and releases folate (Westermarck et. al., 2005). Moreover hypocobalaminaemia may cause decreased folate utilization because cobalamine is needed for conversion of methylfolate to the active form tetrahydrofolate, which is required for DNA synthesis. In addition, an abnormally high numbers of bacteria in the duodenum results in competition for cobalamine, creating a vicious circle (Thompson et al., 2009).

Intestinal dysbiosis, also called bacterial overgrowth (SIBO), is described as an increase in the absolute number of bacteria in the upper SI during the fasting state. Dysbiosis in humans arises secondary to a number of underlying disorders that interfere with intestinal motility, substrate availability and bacteriostatic/cidal secretions. The exactly diagnostic criteria for dysbiosis in companion animals are still debated, nonetheless a genuine bacterial overgrowth exist in conditions equivalent to those in humans (German, 2013). Intestinal dysbiosis may be idiopathic primary (antibiotic responsive diarrhoea) or secondary to an underlying pathology, such as EPI (Thompson et al., 2009). Increased duodenal bacteria have already been reported in cats with EPI (Perry et. al., 1991) and secondary dysbiosis

was likely the cause of the increase folate level in this cat.

Although during secondary dysbiosis appropriate treatment for the underlying pathology is preferable, in some clinical cases concurrent antibacterial therapy is necessary. The choice of the most appropriate antibiotic is controversial, however tylosin and metronidazole are commonly used thanks their spectrum of activity and immunomodulatory activities. Prolonged therapy is often necessary (German, 2013). This cat has been treated with 8 weeks of metronidazole after a first relapse and the prolonged therapy associated to enzyme supplementation led to a resolution of clinical signs.

The association between pancreatitis, IBD and inflammatory hepatic diseases is a common finding in cats (Weiss et. al., 1996) and it is sometimes referred as triaditis, but this terminology is still controversial (Cattin, 2013; Elwood, 2010). The specific conditions of each organ that constitute a diagnosis of triaditis remains to be defined. In fact in actual scientific literature cat triaditis range from any inflammatory process within these organs, to the combination of chronic pancreatitis, chronic cholangitis/cholangiohepatitis and IBD. In any case, the definitive diagnosis of "triaditis" is based on the histopathological evaluation of each organ (Simpson, 2015).

IBD refers to persistent gastro-intestinal signs and histopathologic evidence of chronic intestinal inflammation, causing malabsorption and maldigestion. It is only appropriate to use the term idiopathic IBD if no underlying cause for the inflammation can be found. (German, 2013). Its precise aetiology is still controversial but IBD may be caused by an inappropriate immune response to dietary and/or bacterial antigens presented to the gastrointestinal mucosa (Elwood, 2010). IBD can be further classified based on the predominant inflammatory cells type at histological examination of intestinal biopsies. In this case the patient had a lymphoplasmacytic to neutrophilic enteritis, a quite common histological diagnosis in cats (German, 2013).

In the 80% of cats the major pancreatic and common bile duct form a unique final duct exiting into the duodenum with a common papilla. This particular anatomical feature can allow a reflux of duodenal fluid containing bacteria, bile salts, and activated pancreatic enzymes in both the pancreatic and biliary system, causing inflammation and/or infection (Washabau, 2013). It has also been suggested that an haematogenous spread can be an additional cause of feline inflammatory liver disease (Twedt et al., 2014).

In this case the histology showed a lymphocytic-neutrophilic colangiohepatitis and cholecystitis. Bile culture was also positive for haemolytic *Escherichia coli.* Feline hepatobiliary inflammatory diseases, principally cholangitis, have been generally classified as neutrophilic (acute and chronic), lymphocytic and associated with liver flukes (Twedt et al., 2014), the neutrophilic form being the one most commonly diagnosed at histological examination (Callahan et al., 2011) . In the present case a mixed form of neutrophilic and lymphocytic cholangiohepatitis was diagnosed and for this reason the patient received a

combined therapy with antibiotics and steroids.

The exact aetiology of pancreatitis in cats is still not clear and there are a number of potential triggers of pancreas inflammation (enteric bacteria, immune-mediated diseases, idiosyncratic drug reactions, diet, idiopathic causes and others). It is not even precisely known if pancreatitis is caused by enteritis/colangiohepatitis or if it may be the initiating stimulus for triaditis (Simpson, 2015). Histopathologic criteria for the classification of pancreatitis have not been universally standardized in veterinary medicine and different histopathologic scoring systems have been proposed for both cats and dogs (De Cock et al, 2007; Hess et al, 1998). Acute pancreatitis ranges from oedematous to necrotising and it can progress to chronic pancreatitis characterized by fibrosis, inflammation and atrophy (De Cock et al, 2007), and then to exocrine pancreatic insufficiency (Simpson, 2015).The histopathology of the pancreatic samples of this cat showed severe chronic pancreatitis and a diffuse loss of acinar cells replaced by areas of fibrosis. It is then likely that chronic pancreatitis caused a progressive loss of exocrine pancreatic cells, leading to a lack of pancreatic enzymes and development of typical clinical signs of EPI.

LEARNING POINTS/TAKE HOME

- EPI is rare in cats and the most common cause is chronic pancreatitis
- Typical clinical signs in cats with EPI are weight loss, polyphagia, clay-coloured abundant stools and greasy-looking coat on the hind limbs, perianal and tail regions.
- Therapy for concurrent pancreatitis, inflammatory bowel disease and cholangiohepatitis cats may include a combination of antibiotic, immune-suppressive drugs and diet change. Therapy for EPI consists in life-long supplementation of pancreatic enzymes.
- Cats with EPI have frequently low level of vitamin B12, therefore cobalamine should be supplemented by monthly sub-cutaneous injection.
- Intestinal dysbiosis is a common consequence of EPI and appropriate prolonged antibiotic therapy is often required.

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FIGURE/VIDEO CAPTIONS

Figure 1. Greasy and wet looking of the coat
Figure 2. Clay-coloured appearance of the stools
Figure 3. Right – lateral X-ray of the thorax with enlarged sternal lymph nodes
Figure 4. Right – lateral X-ray of the abdomen, diffuse loss of serosal detail
Figure 5. Abdominal ultrasound: thickened gallbladder wall and tortuous intra and extra hepatic biliary ways
Figure 6. Abdominal ultrasound: increased thickness of the small intestinal muscularis and hypoechoic mucosa
Figure 7. Abdominal ultrasound: mesenteric lymphadenopathy and free peritoneal fluid
Video 1. Polyphagia

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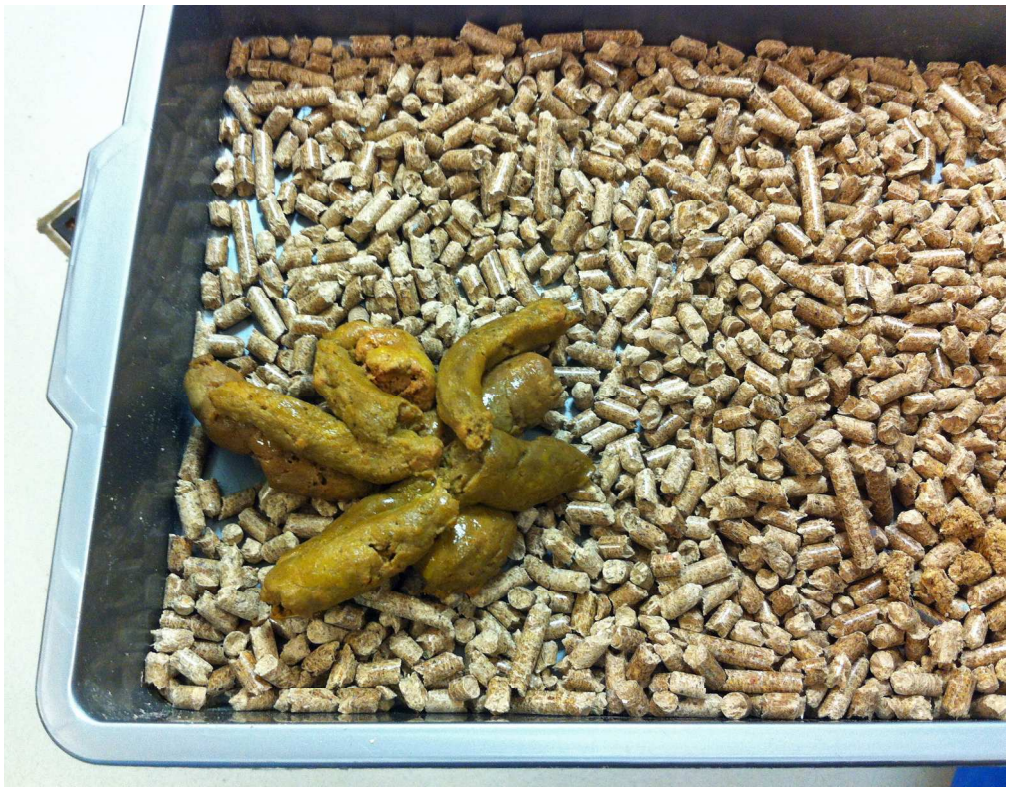

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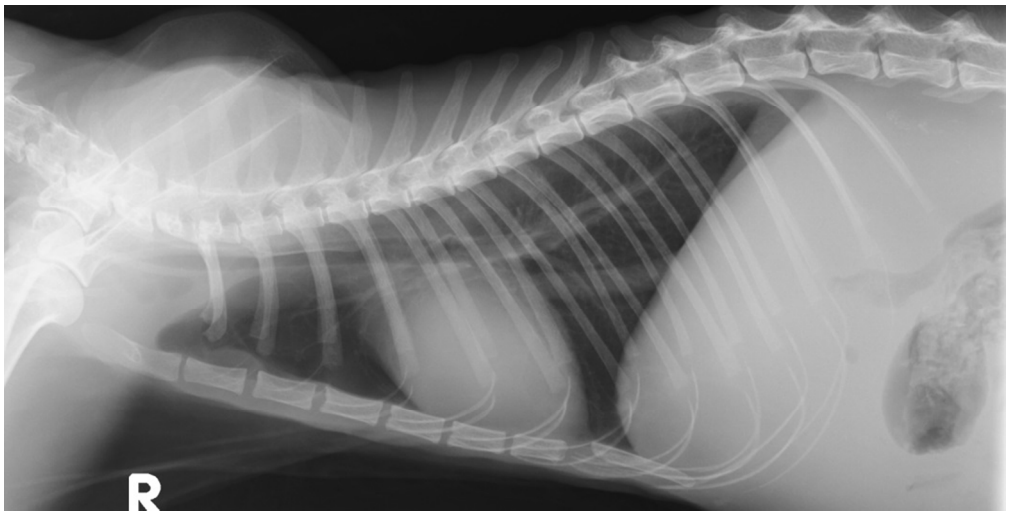

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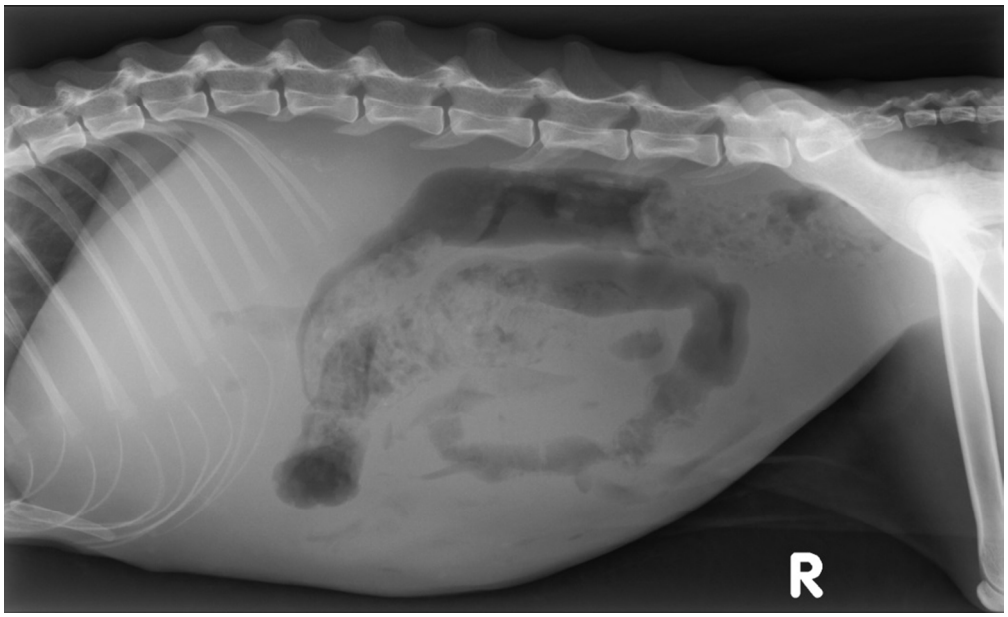

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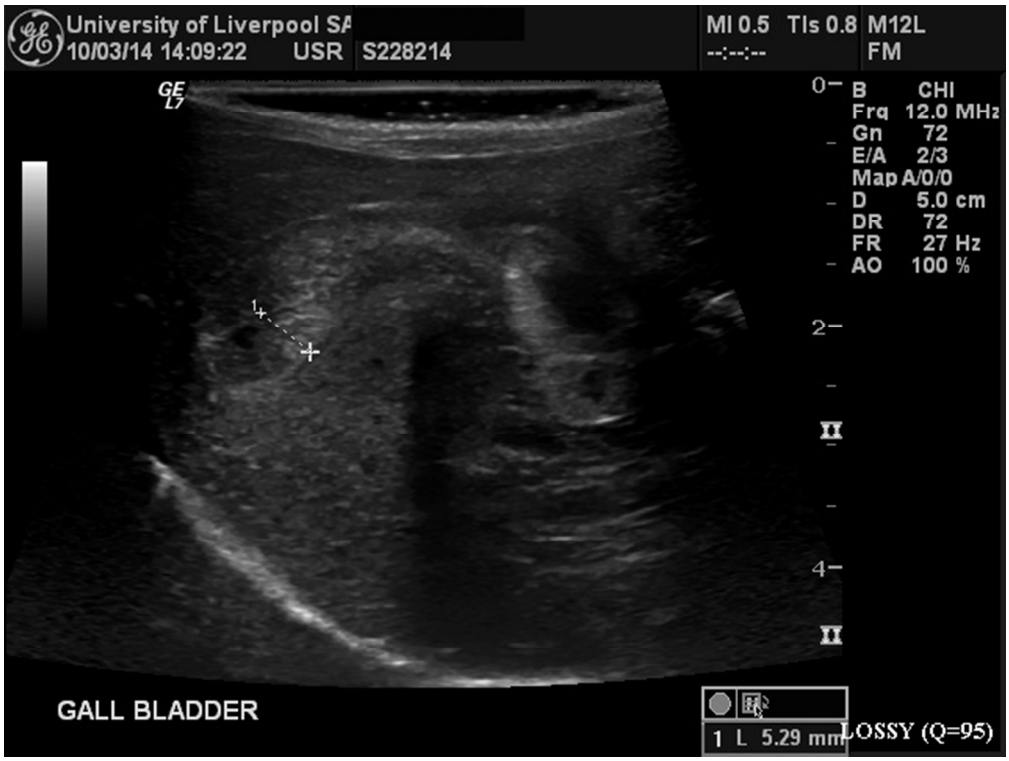

Ultrasounds picture of gallbladder
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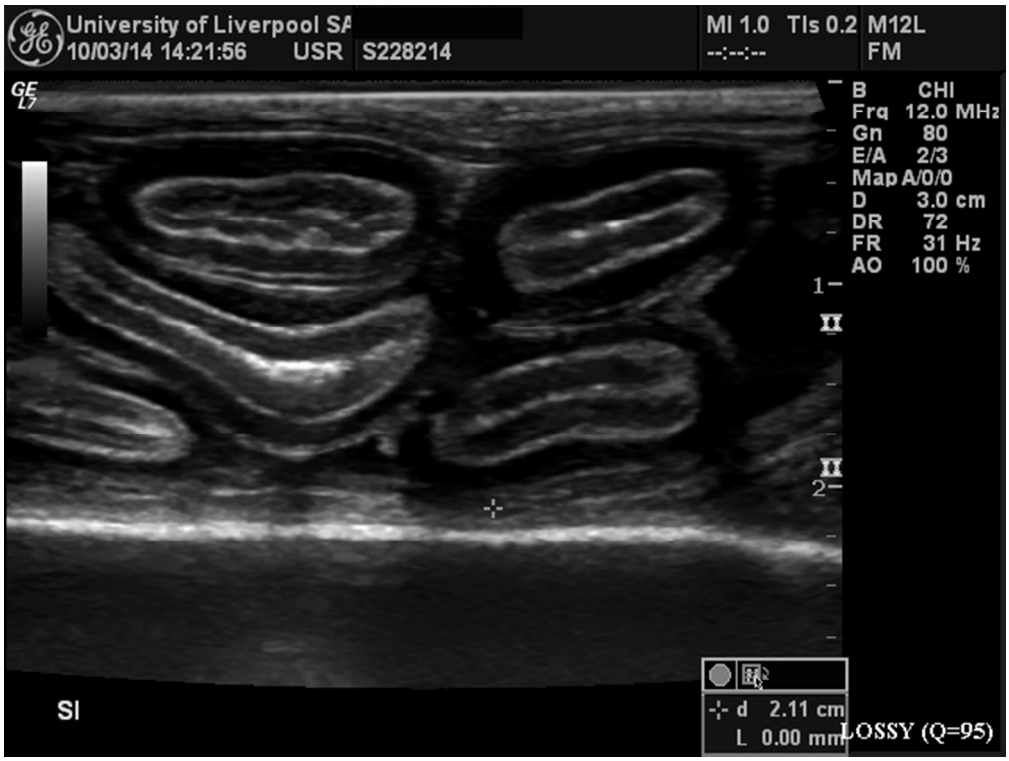

Ultrasound pictures of small intestines
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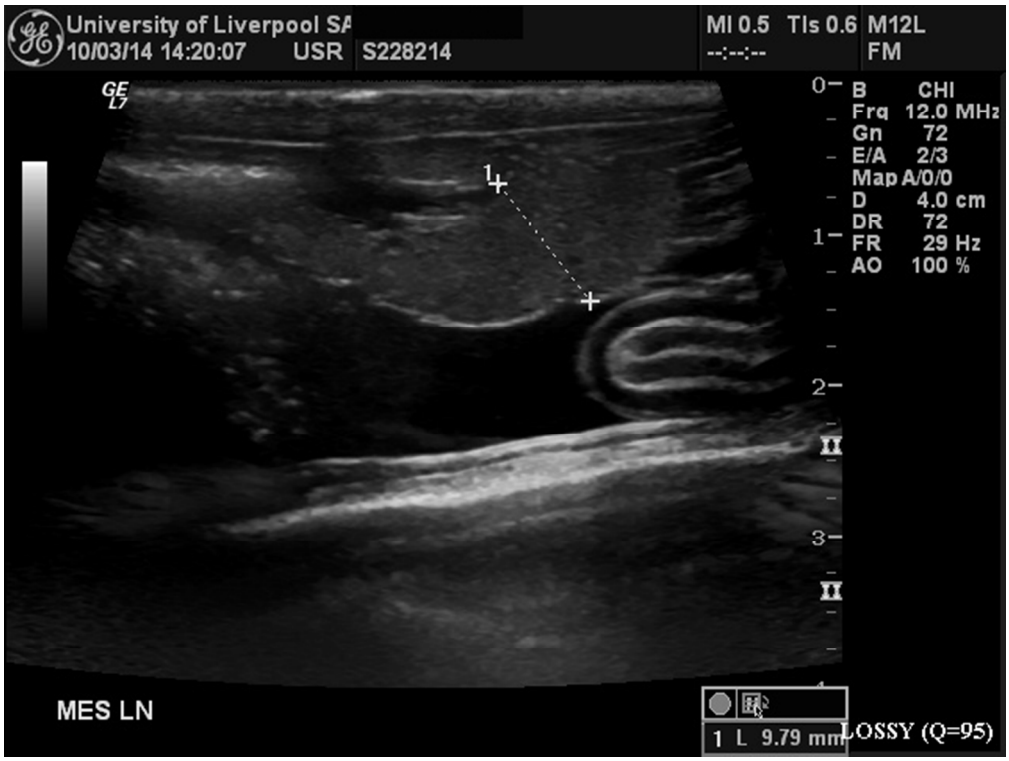

Ultrasounds picture of abdominal fluids
225x169mm (72 x 72 DPI)