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FELINE INFECTIOUS PERITONITIS, TWO DIFFERENT MANIFESTATIONS OF THE EFFUSIVE FORM. CASE REPORT

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ABSTRACT

Feline infectious peritonitis (FIP) is a systemic, potentially fatal viral disease of domestic and wild felids. This study demonstrates the pleural and abdominal manifestation of the effusive form of FIP from the clinical and pathological points of view. Two approximately one-year-old cats (male and female) are presented. A set of clinical (haematology, biochemistry), morphological (cytology, necropsy histopathology and immunohistochemistry) and other diagnostic assays (PCR, FIP antibodies detection, protein electrophoresis) were carried out. The results, like lymphopenia, lower A/G ratio, and hyperbilirubinemia are among the most characteristic signs for FIP. The morphology revealed the occurrence of effusions, severe diffuse adhesive pleuritis, icterus, vasculitis/perivasculitis, severe liver and lungs alteration and granulomatous-like reactions with the presence of giant cell type macrophages in the lungs. At the end of this work a list of tests required for FIP diagnosis is mentioned.

Key words: case report; cats; FIP; summary of diagnostic tests

INTRODUCTION

Feline infectious peritonitis is a viral disease of cats that occurs throughout the world. FIP is caused by the feline infectious peritonitis virus (FIPV) which is one of the biotypes of feline coronavirus (FCoV). The second is feline enteric coronavirus (FECV) [5].

According to the disease characteristics, FIP can be divided in two types: effusive and non-effusive [20]. The effusive or "wet" form of the disease is also known as a wet peritonitis and its main symptom is abdominal swelling. If the cat has pleural effusion it may also present with dyspnoea and lung noise on auscultation [10].

Clinical signs of FIP vary depending on organ involvement. Many organs, including the liver, lungs, kidneys, pancreas, CNS and eyes can also be involved [11, 17].

Diagnosis of FIP remains a combination of history and clinical signs along with a variety of clinical assays.

Histopathological examination and immunohistochemical (IHC) presentation of FCoV antigens, which are usually localised around a lesion, are accepted as the gold standard for a definitive diagnosis [2].

This paper deals with the pleural and abdominal manifestations of the effusive type of FIP from the clinical and pathological points of view. Two approximately one-year-old cats (male and female) were presented at two different veterinarian institutions which have different possibilities for the implementing a series of diagnostic tests. Nevertheless, we decided to include both cases in our study, in order to highlight the need for a comprehensive approach to diagnosis. This is why, at the end of this paper, we have provided an overview of the tests that should always be used in the context of an FIP diagnosis.

CASE PRESENTATION

A one-year-old, bilateral cryptorchidic European shorthaired male cat adopted from the street was presented due to problems with walking lasting approximately two days. On the distal end of the right thoracic limb, in the area of the central pad, a painful bite wound (*vulnus morsum*), with a purulent secretion came out upon compression. After the treatment, the pad was almost healed and painless within the next 2–3 days.

Over the next few days, the cat's health worsened, manifested by impaired breathing, it did not take food or liquids, and the body temperature was 40.5 °C.

Haematology (carried out on a Heska Element HT5) showed a stress leukogram, including lymphopenia (0.33 10^9 l^{-1}; ref. 0.73–7.86) and mild eosinopenia (0.33 10^9 l^{-1}; ref. 0.06–1.93). A decrease in lymphocytes (LYM) (0.034 %; ref. 0.120–0.450) and eosinophils (EOS) (0.004 %; ref. 0.010–0.110) was also seen and an increase in neutrophils (NEU) (0.913 %; ref. 0.380–0.800), as well.

The biochemical parameters (obtained from SEAMATY biochemical analyser) were within normal limits, except for the albumin/globulin ratio, which was 0.7.

USG examination (carried out using a Mindray DP 50Vet) revealed the presence of fluid on the left side of the body close to the diaphragm, behind the liver. Hydrothorax was also present with well seen fibrinous attachments between the *apex cordis* and the diaphragm. Severe hypertrophy of the left myocardial ventricle was also observed. This examination was very painful for the cat. Finally, the suspected diagnosis of FIP and hypertrophic cardiomyopathy was accepted.

MANAGEMENT AND OUTCOMES

Infusion therapy (NaCl 0.9 % Braun solution, B. Braun Medical S.A., Rubi, Barcelona, Spain), at a rate of 5 ml per hour for 24 hours with an admixture of rehydration solution of Duphalyte inj. (Zoetis s.r.o., Czech Republic), with a complex composition, containing vitamins, electrolytes, amino acids and nutritional components, in a dose of 10 $ml.kg^{-1}$ per 24 hours, administered automatically using an infusion pump), oxygen therapy, and diagnostic and therapeutic thoracentesis were carried out. The aspiration of approximately 70 ml of a viscous, straw yellow liquid, with a specific gravity of 1035, pH 7, protein content +++, Leuk 1, Hb+, Gluc+ was performed. The cytology revealed a high content of macrophages, but the presence of neutrophils and lymphocytes was minimal.

After initiation of therapy, no any positive clinical changes were evident, and also because of the financial possibilities of the owner, euthanasia was performed at her request (introduction to general anaesthesia with a combination of: butorphanol in the preparation of Butomidor 10 $mg.ml^{-1}$ inj. (VetViva Richter GmbH, Durisolstrasse 14, Wels 4600, Austria) at a dose of 0.4 $mg.kg^{-1}$ i.v.; medetomidine in the preparation Cepetor 1 $mg.ml^{-1}$ inj. (CP-Pharma Handelsges. MbH, Ostlandring 13, 31303 Burgdorf, Germany) at a dose of 0.01 $mg.kg^{-1}$ i.v.; midazolam in the preparation Midazolam Accord 5 $mg.ml^{-1}$ inj. (Accord Healthcare Polska Sp. z. o.o., ul. Taśmowa 7, 02-677 Warsaw, Poland) at a dose of 0.3 $mg.kg^{-1}$ i.v.; ketamine in the preparation Ketamidor 100 $mg.ml^{-1}$ inj. (Richter Pharma AG, 4600 Wels, Austria) at a dose of 3 $mg.kg^{-1}$ i.v.). When the patient was under proper general anaesthesia, we proceeded to euthanasia with sodium pentobarbital in Exagon 400 $mg.ml^{-1}$ inj. (VetViva Richter GmbH, Durisolstrasse 14, Wels 4600, Austria) at a dose of 5 ml for this i.v., slowly.

Consequently, the necropsy was carried out. Mild focal fibrinous perihepatitis was found in the abdominal cavity. Hydrothorax (approximately 80 ml of serous fluid), severe diffuse fibrinous adhesive pleuritis (Fig. 1), concentric myocardial hypertrophy of the left ventricle and bilat-

eral scrotal cryptorchidism were found. Small, whitish, and randomly distributed nodules were seen in the caudal lobes of lungs.

Histology revealed the presence of diffuse infiltration and the thickening of the alveolar walls by mononuclear cells, accompanied by haemorrhages. Severe alteration of the lungs parenchyma, noticed due to the multiply necrotic foci and diffuse pattern of lympho-epithelioid granulomatous-like reactions with the presence of multinucleated macrophages, was also found (Fig. 2). In the myocardium oedema, perivasculitis and disarray of cardiomyocytes with the poor lymphocytic infiltration were observed. In the liver, severe hyperaemia and haemorrhages, and in the spleen pyknosis of some lymphocytes were detected, as well. The IHC examination for FIP-virus antigen detection from lungs tissue was positive (Laboklin Slovakia Ltd.) (Fig. 3).

CASE PRESENTATION

A seven-months-old female, non-sterilised, vaccinated kitten was presented with a medical history of 5 days of apathy, inappetence and fever. The owners observed abdominal enlargement. On admission, cachexia, a moderate degree of dehydration, apathy, weakness, abdominal distention, anaemic mucous membranes, slightly icteric skin in the abdominal area, and bilateral cardiac murmur were observed.

Haematology revealed eosinopenia (0.01 10^9 l^{-1}; ref. 0.17–1.57), lymphocytosis (9.48 10^9 l^{-1}; ref. 0.92–6.88) and not severely decreased values of haematocrit (HCT) (29.1 %; ref. 30.3–52.3), haemoglobin (HGB) (9.4 $g.dl^{-1}$; ref. 9.8–16.2), mean corpuscular volume (MCV) (31.1 fl; ref. 35.9–53.1) and mean corpuscular haemoglobin (MCH) 10.0 pg; ref. 11.8–17.3). Insignificant increase in the values of red blood cell distribution width (RDW) 34.6 %; ref. 15.0–27.0), reticulocytes (53.4 $K.\mu l^{-1}$; ref. 3.0–50.0) and mean platelet volume (MPV) (22.4 fl; ref. 11.4–21.6) were detected, as well.

All biochemical parameters except total proteins (66.1 $g.l^{-1}$; ref. 57–85) were changed. The data are summarised in Tab. 1.

Tab. 1. Results of biochemical examination

Parameter	Results	Units	Reference interval
Creatinine	55.3	$\mu mol.l^{-1}$	59–168
Urea	19.90	$mmol.l^{-1}$	8.2–12.1
Albumin	18.2	$g.l^{-1}$	24–85
AST	473.39	$U.l^{-1}$	< 58
Total bilirubin	71.47	$\mu mol.l^{-1}$	< 3.4
ALT	236.05	$U.l^{-1}$	< 99
SDMA	0.89	$\mu mol.l^{-1}$	< 0.75

AST – aspartate transaminase; ALT – alanine transaminase; SDMA – symmetric diethylarginine

To confirm the diagnosis, further examinations were made. A summary of them is given in Tab 2. They were performed in Laboklin Slovakia Ltd, which provides clinical diagnostic tests for veterinary surgeries.

The Rivalta test done according Tasker [15] gave a positive reaction.

USG examination demonstrated the effusion in abdominal cavity close to the left kidney.

MANAGEMENT AND OUTCOMES

Infusion and symptomatic therapy were initiated during daily hospitalisation. The owners administrated the antiviral drug GS441524 at their own discretion. The kitty

Tab. 2. Summary of further diagnostic tests (Laboklin Slovakia Ltd.)

Parameter	Conclusion	Result	Units	Reference interval
Coronavirus PCR quantitative	positive (high concentration)	30 (coronavirus content)	Million per 1 ml aspirate	
FIP antibodies	positive	31.04	LE	< 9
Protein electrophoresis				
Albumin globulin ratio	low (positive)	0.30		> 0.6
Albumin	low	22.8	%	42–61
Alfa - globulin	high	29.8	%	9–24

FIP – feline infectious peritonitis

was in treatment for 5 days. Infusion therapy was initiated immediately using rehydration solutions: Natrii chloridum 0.9 % sol. inf. (B. Braun, Germany), Glucose 5 % $w.v^{-1}$ intravenous infusion sol. inf. (B.P. Bieffe, Italy), Duphalyte inj. (Zoetis, Czechia). To stimulate the appetite, the vitamin B12 containing preparation Catosal 10 % ($w.v^{-1}$) inj. (Bayer, Germany) was administrated. Preventive antimicrobial therapy was provided using the antibiotic Synulox RTU 140/35 $mg.ml^{-1}$ inj. (Zoetis, Czechia). The anti-vomiting drug Degan 10 mg.2 ml^{-1} sol. inj. (Sandoz Pharmaceuticals d.d., Slovenia), the analgesic Butomidor 10 $mg.ml^{-1}$ inj. (Richter Pharma AG, Austria) and the hepatoprotective Hepagen 100 $mg.ml^{-1}$ inj. (Fatro S.p.A., Italy) were also administered. After the deterioration of the medical condition, we proceeded to the application of the corticosteroid Colvasone 2 $mg.ml^{-1}$ inj. (Norbrook, Northern Ireland). Due to dyspnoea and pulmonary oedema, Furosemide BBP 10 $mg.ml^{-1}$ inj. (BB Pharma a.s., Czechia) was administered intravenously. During hospitalisation, Nutribound cats 150 ml (VIYO International N.V., Belgium) was administered *per os*.

Gradual deterioration of the health condition occurred. Icterus, abdominal distension, and inappetence became more pronounced. On the fifth day, the cat decompensated with worsening dyspnoea. A prominent cardiac murmur was present bilaterally, with additional murmurs (grunts) audible in the lung field. Control by ultrasound (Aloka prosound Alpha 6 Diagnostic ultrasound system) examination showed the presence of fluid both abdominally and pericardially. Partial therapeutic abdominocenthesis was performed in order to stabilise the patient. Oxygen therapy was also provided. Despite the therapy, the patient spontaneously died.

Necropsy revealed generalised icterus, ascites (approximately 70 ml serosanguinous fluid), fibrinous peritonitis oedema of the lungs, eccentric hypertrophy of the left ventricle and dilatation of the right ventricle.

Histology found severe alteration of liver with the occurrence of multiply necrotic foci occurrence, accompanied by mononuclear cell infiltration; vasculitis and perivasculitis were also found. In the lungs the thickening of alveolar walls by mononuclear cells, oedema and vasculitis were seen. Severe tubulonephrosis was also observed in the cortical part of the kidneys, and oedema and perivascularitis were seen in the myocardium (Fig. 4).

DISCUSSION

Feline infectious peritonitis (FIP) is one of the most important infectious diseases and causes of death in cats. Young cats less than 2 years of age are especially vulnerable [13]. In this work two cases of the effusive type of FIP with different clinical and pathological lesions are described.

Haematological abnormalities are very common in cats with FIP. Lymphopenia is known to be present in about 50 % of FIP positive cats with effusion [4] and in combination with neutrophilia it is known as a "stress leukogram" [1], which was found in our first case. In contrast, in the second case, aside from anaemia, severe lymphocytosis was observed. This phenomenon was found in FIP-infected cats during treatment with the anti-viral drug GS-441524 [19] which was applied also in our case. Since the severity of initial lymphopenia is considered a negative prognostic factor, lymphocytosis during the treatment might indeed be a positive prognostic factor [19]. In our case, the antiviral drug application was not successful. In this regard, it should be noted that there are two main hypotheses concerning FIP pathogenesis. In both of them, the key pathogenic event is the massive replication of the virus in macrophages. If the cat does not eliminate macrophages infected with replication-competent virus early in the infection, the presence of virus within circulating macrophages initiates an ultimately fatal arthus-type immune-mediated reaction [17]. We do not have an information about when exactly, in which phase of diseases, the owners started to administer this drug. We assume the virus was already present within circulating macrophages and initiated the fatal outcome of the disease. There are some other factors that increase the virus replication, like young age, stress and corticosteroid therapy [20], all of them present in our case. It remains of particular interest to further determine the immunological processes and cellular immune response against the virus before, during and after treatment [19].

The vast majority of cats with FIP also have abnormalities upon serum biochemistry. The most important parameter is the A/G ratio (in this study 0.7 and 0.3, respectively). Its reduction occurs in almost all cases of FIP (40–90 %) [3]. Different studies have shown different results on the effective critical value of the A/G ratio. According to results in a retrospective study of 127 cats, a value 0.5 could be considered critical as highly susceptive of FIP. When

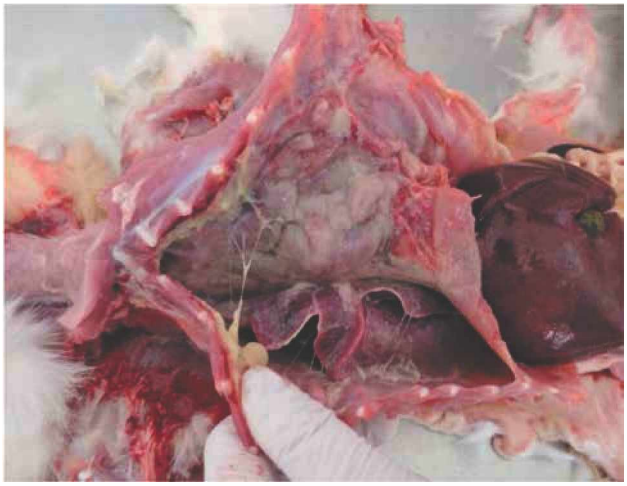

Fig. 1. Severe diffuse fibrinous adhesive pleuritis found in an one-year old European short-haired male cat (the first case)

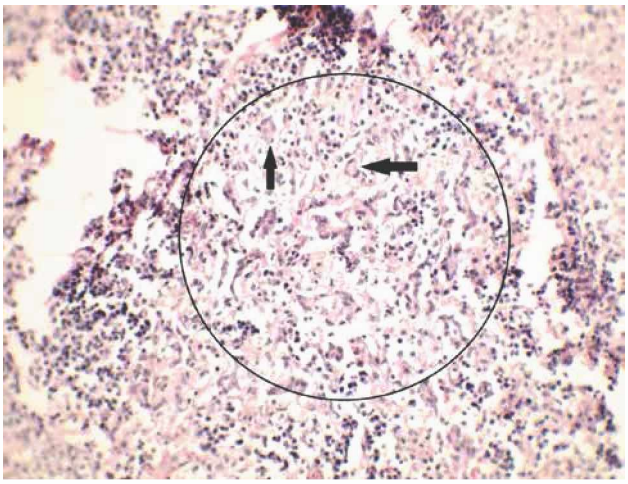

Fig. 2. A diffuse pattern of lympho-epithelioid granulomatous reaction (in a circle) with the presence of multinucleated macrophages (arrows) was observed in the lungs of the cat in the first case. Haematoxylin and eosin. Magn. x 20

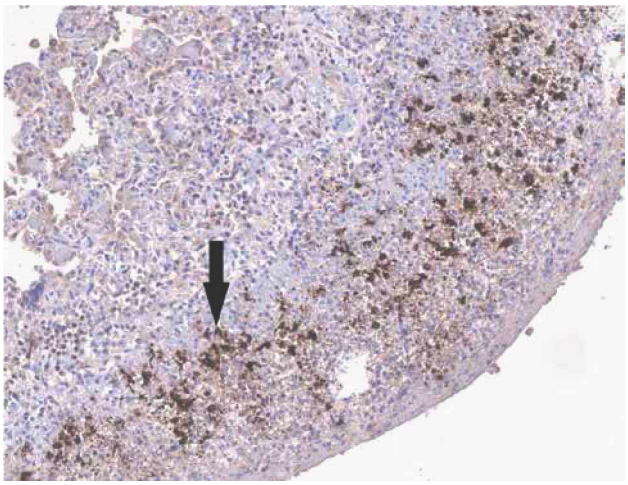

Fig. 3. The IHC examination for FIP-virus antigen detection (arrow) from fibrinous pleuritis. Multiple positive reactions (one of them at the tip of the arrow) can be observed. IHC (FIP-virus antigen), Magn. x 10.
Laboklin Slovakia Ltd.

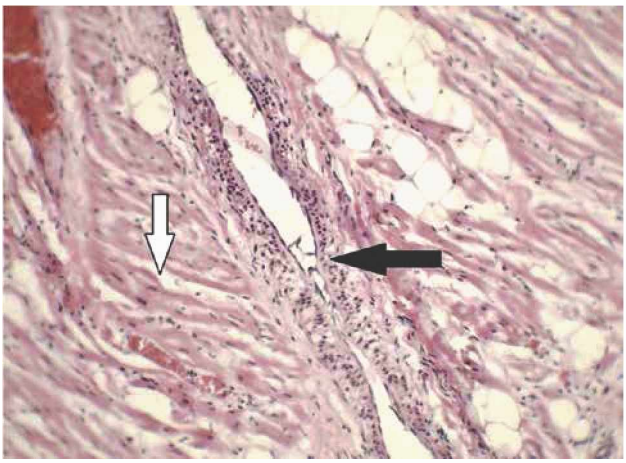

Fig. 4. In both cases myocardial oedema (white arrow) and perivasculitis (black arrow) were detected. Haematoxylin and eosin. Magn. x 20.

the A/G ratio > 0.8, the possibility of FIP is very small; when 0.5 < A/G ratio < 0.8, FIP remained a possibility; when the A/G ratio < 0.5, FIP was highly suspected [20].

Almost 46 % of effusive FIP-diseased cats exhibited hyperbilirubinemia at first presentation in comparison to 100 % before death. This increase in bilirubin levels is due to destruction of the red blood cells and the accumulation of haemoglobin that often accompanies FIP [7]. In our second case, some other changes, included in the aetiology of this condition, like hepatic necrosis with a dramatic increase in liver enzymes (ALT and AST), were found. It is thought that hyperbilirubinemia serves as an indicator for diseases stage, especially in the effusive abdominal form [14].

The variability of the haematological and biochemical results detected in our work correlates with the knowledge that these parameters are neither pathognomonic nor specific for FIP on their own, meaning that they can occur in any cat suffering from various diseases with an inflammatory basis, and this should be looked for to support a diagnosis of FIP [15].

From a clinical presentation and/or morphological point of view, the most typical change in cats with FIP is effusion. In one study of 224 cats with confirmed FIP, 78 % of the cats had effusions. Typically, this is a cellular-, protein- and fibrin-rich modified transudate [8], which is consistent with our evaluation. Macroscopic and cytological examination of these effusions are important in order to exclude or confirm other differential diagnoses (such as lymphoma or bacterial peritonitis or pleuritis) [4].

The classical effusion of wet FIP results mainly from acute damage to the vessel walls and leakage of plasma into the interstitial spaces and eventually into body cav-

ities. This can occur because activated monocytes spread the virus, attach to blood vessels and cause the formation of focal infiltrates in the blood vessel walls [9]. Vasculitis or perivasculitis is almost the most specific microscopic lesion of the disease. It is observed in small and medium-sized venules in affected tissues and organs [11], including lungs, myocardium and liver, as was found in both our cases.

The thoracic manifestations of FIP are less common than the abdominal ones. Only one case [2], which reports the lungs lesions presented by mononuclear infiltration in a diffuse manner and a thickening of alveolar walls by these cells, was identical to ours. In a study dealing with all respiratory lesions in FIP, major histological patterns in different combinations were defined as pleuritis, vasculitis, perivascular injury and pneumonia [11]. Foci of necrosis in pleura and lungs were also detected. This finding is partially consistent with our observations, under which severe alteration of lung parenchyma presented by multiply necrotic foci and diffuse lympho-epithelioid granulomatous–like reactions, with the presence of multinucleated macrophages, was found. The granulomatous reactions are known as macrophage-dominated lesions, and these partially isolate the healthy tissue from the irritant and the irritant-induced inflammation [6]. They can occur in any organ [18], but the pulmonary system is one of the most commonly affected sites to encounter granulomatous inflammation [12]. FIP, as well as some bacteria and fungi, may trigger these reactions in cats. This may be due to long-lasting cell-mediated immune reactions. Although cell-mediated immune response effectively fight of these infections in most cats, in a few animals the immune response is only partially effective and results in a mass at the site of infection [18].

CONCLUSIONS

This study described two cases of FIP effusive forms. The results showed great variability. Some of them, like lymphopenia, lower A/G ratio, and hyperbilirubinemia is among to the most characteristic signs for FIP. Morphology revealed the occurrence of effusions, severe diffuse adhesive pleuritis, icterus, vasculitis/perivasculitis, severe liver and lungs alteration and granulomatous reaction with the presence of giant cell type macrophages in the lungs. Consequently, to arrive at a diagnosis of FIP, the veterinarian must consider the individual patient's history, signalment and physical examination findings. To be successful in this diagnostic work it is recommended to follow a systematic overview [13, 16] which includes diagnostic tests from:

Blood: 1. Complete blood count (CBC) 2. °Serum biochemistry (globulins, albumins, bilirubin, A/G ratio); 3. Alpha-1-acid glycoprotein (AGP – good biomarker for inflammation and liver disease).

Effusion: 1. The Rivalta test; 2. Cell count and cytology; 3. Bacterial culture; 4. Biochemical analysis – protein and A/G ratio.

Cerebrospinal fluid (CSF): 1. Cell count and cytology; 2. Protein concentration.

Aqueous humour: cell count and cytology.

Other: 1. Routine diagnostic imaging (RTG and USG) 2. Advanced diagnostic imaging (CT or MRI).

Cytology of effusions.

Histopathology of tissue samples (e.g. liver, kidney or mesenteric lymph nodes) collected ante-mortem or at post-mortem.

Immunohistochemical/cytochemical examinations of histological or effusion cytology samples, respectively.

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