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CLINICAL CASE

Unusual clinical presentation of leptospirosis in a cat☆

Présentation clinique originale d'une leptospirose chez un chat

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Azotemia;
Cat

Summary A sterilised 4-year-old cat, living in contact with hunting dogs, was presented following several days of vomiting and diarrhea. In a state of shock, she was hyperesthetic and handling was painful. She had marked inflammatory lesions on her pinnas, abdominal skin and foot digits. Proteinuria was elevated without significant sediment. Blood analysis revealed pre-renal azotemia, and hyperglobulinemia. An abdominal ultrasound examination showed nephromegaly and an echomodified pancreas (snap fPL test negative). A search for anti-nuclear factors was negative. PCR blood analyses were slightly positive for *Leptospira* spp. and negative for *Ehrlichia* spp. and *Anaplasma* spp. A treatment based on cefovecin and methylprednisolone (5 days) then 14 days afterwards, doxycycline (4 weeks) was prescribed. The cat recovered completely. A double serology test, carried out at 5 weeks' interval, showed a significant seroconversion for Leptospira serogroup Sejroë serovar Saxkoebing (snap FeLV/FIV combo test negative). This case shows that cats can develop clinical leptospirosis without an apparent cause of immunosuppression, with symptoms partly comparable to those of the canine subacute form, and particularities. The Sejroë serogroup will therefore have to be included in the future in serological testing if there is a clinical suspicion of leptospirosis in cats.

☆ Crédits de formation continue. — La lecture de cet article ouvre droit à 0,05 CFC. La déclaration de lecture, individuelle et volontaire, est à effectuer auprès du CNVFCC (cf. sommaire).

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MOTS CLÉS
Chat ;
Leptospirose subaiguë ;
Vasculite ;
Glomérulonéphrite ;
Insuffisance rénale

Résumé Une chatte stérilisée de 4 ans, en contact avec des chiens de chasse, est présentée pour vomissements et diarrhée depuis quelques jours, en état de choc, hyperesthésique et douloureuse à toute manipulation, avec des lésions inflammatoires marquées du pavillon des oreilles, de la peau de l'abdomen et des doigts. Les analyses révèlent une protéinurie importante sans culot significatif, une azotémie prérénale et une hyperglobulinémie. L'échographie abdominale montre une néphromégalie et un pancréas d'échodensité modifiée (test snap fPL négatif). Le dosage des facteurs antinucléaires est négatif et les analyses PCR sur sang faiblement positives pour *Leptospira* spp. (négatives pour *Ehrlichia* spp. et *Anaplasma* spp.). Un traitement à base de céfovécine et méthylprednisolone (5 jours) puis 14, après, de doxycycline (4 semaines) est prescrit. La chatte récupère complètement. Une sérologie double, à 5 semaines d'intervalle, montre une séroconversion significative pour Leptospira sérogroupe Sejroë, sérovar Saxkoebing (test snap combo FelV et FIV négatif). Ce cas montre que les chats peuvent développer une leptospirose clinique sans cause apparente d'immunosuppression, avec des symptômes comparables à ceux de la forme subaiguë du chien et des particularités ; le sérogroupe Sejroë devra donc être inclus dorénavant dans les recherches sérologiques en cas de suspicion clinique chez le chat.

Introduction

Leptospirosis is a serious wide spread illness which affects many mammals. It is a potential zoonotic infection, which causes real problems of public health, because it can be transmitted by droplets of urine in direct contact with normal mucous membranes or damaged skin [1].

Cats are sometimes seropositive with prevalence ranging from 4.8 to 35% and dominant serogroups can vary according to geographical location and the feline population studied [2—4]. Still as regards cats, some natural seroconversions have been described [5]. Cases of leptospiruria have been revealed using PCR [2,6], but very few symptoms have appeared in response to experimental infections (1 °C temperature increase, rare appearance of poluyuro-polydipsia [PU/PD], with no mortality [2,7]). There are only very few described cases of clinical leptospirosis in cats [5,8—13]. The symptoms described were most frequently PU/PD and temperature, sometimes accompanied by jaundice and the mortality rate described was high. In the present case, the clinical symptoms were notably different from those described previously and the outcome was favourable.

Observation

Medical history

A European cat called Mimi, 4 years old and sterilised three months previously, was brought into the clinic because she had been vomiting and losing weight over the previous few days. This cat, which formerly lived in a flat, had been going outside for 6 months and lived in contact with hunting dogs. She had not been vaccinated nor regularly wormed nor treated for external parasites.

Clinical examination

When clinically examined all over, the cat appeared to have lost a lot of weight (BMI 3/9), with dull fur in a bad state. She had a marked redness on her pinnas. She was tachypneic (at 70 bpm), hyperesthetic and even allodynic. Closer clinical examination showed dehydration with a persistent skin fold and sticky mucous membranes of normal colour as well as hypothermia (37.6 °C). Mimi had a left parasternal systolic murmur (2/6) and tachycardia (220 bpm). Her popliteus and inguinal lymph nodes were enlarged and her skin was swollen, with hyperemia, hot and painful (pinnas, foot digits and abdomen with a marbled appearance, Fig. 1). When pressure was applied to the pinna or the skin of the belly, with a pane of glass, the hyperemia disappeared and reappeared very quickly when the pressure was relieved. A proteinuria reading of 3+ was observed without any other anomaly showing on the strip and without significant sediment (urine density reading using a refractometer: 1.050).

Diagnostic hypotheses

The state of shock could be due to kidney failure, a third sector, an electrolyte disorder, systemic inflammatory response syndrome (SIRS), sepsis or cardiac decompensation, pancreatitis or an autoimmune disease affecting internal organs (lupus). Skin lesions could be explained by SIRS, an immune complex reaction or an autoimmune illness, a reaction to drugs (patient's medical history not in favour of this), sepsis or vasculitis.

Complementary examinations

Biochemical blood analysis showed moderate hyperglycemia (2.13 g/L), pre-renal uremia (1.14 g/L, UV < 0.76) and creatinemia (14.9 mg/L, UV < 24) as well as ALT (16 U/L) and ALKP (13 U/L) liver enzymes within normal range. The Alb/Glob ratio was 0.52 (26/50; UV > 0.8). A snap text (Idexx) did not reveal a serum increase in feline pancreas specific lipase.

The blood count showed neither neutophilia nor thrombopenia. The platelets had on the other hand a systematically active appearance in a blood smear, with many

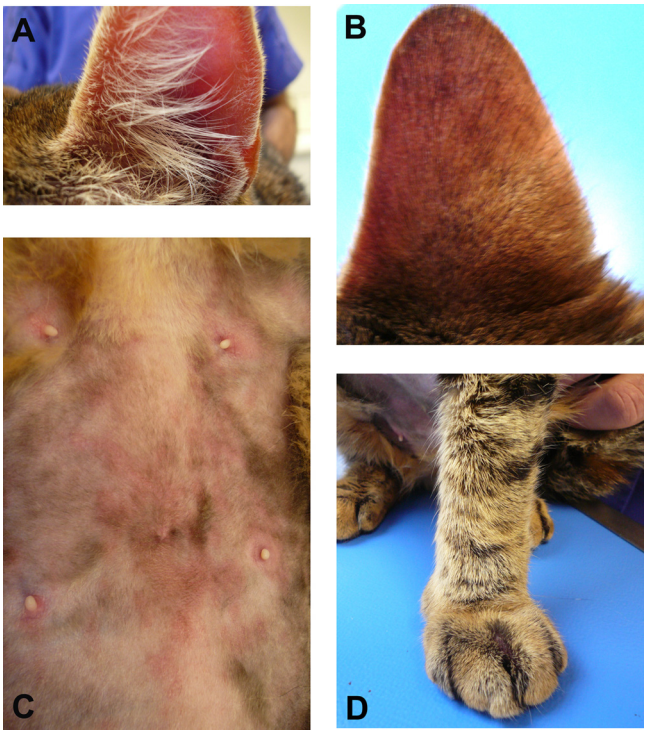

Figure 1. General appearance of the cat at the first consultation. The pinnas (A and B) and abdomen skin (C) were swollen, hot and painful, with hyperemia of marbled appearance that disappeared and reappeared very quickly after pressure. Foot digits were swollen and congested as well (D).

cases of platelet evagination, the red cells appearing normal (hemoglobinemia at 9.6 g/dL, UV > 9.5).

The abdominal ultrasound examination (Fig. 2) showed a heterogenous liver parenchyma, which is hyperechogenic in comparison with the spleen. The pancreas was visible with heterogenous echogenicity (a hypoechogenic centre and a hyperechogenic perimeter). The two kidneys had a normal echo structure with a renal cortex, which was slightly hyperechogenic and nephromegaly (4.3 and 4.7 cm). The rest of the examination was normal. No sign of effusion (neither pleural nor abdominal) was revealed.

When faced with glomerulopathy or a tubulopathy (proteinuria without significant sediment and without glycosuria), marked skin inflammation of extremities resembling vasculitis and a decrease in the albumin over globulin ratio (inflammatory phenomena), an infectious illness involving Erhlichia, Anaplasma or Leptospiras, or disseminated lupus erythematous was suspected. The quantity of serum antinuclear antibodies was requested as well as a polymerase chain reaction on whole blood (EDTA) and urine to carry out a DNA search for suspect pathogenic agents.

The blood PCR came back slightly positive for *Leptospira* spp. (infectious nucleic acid in a low concentration in the blood, absent in urines). Other analyses were negative. Clinical leptospirosis was therefore suspected.

Treatment

The cat was immediately put on a drip (Ringer's lactate) to deal with the tissue hypoperfusion and treated with maropitant (1 mg/kg IV/d, 2 d) to deal with the vomiting, then, whilst waiting for results, doxycycline 24 hours after admission (50 mg/d PO). Seventy-two hours after admission, the cat's temperature was 39.0 °C. The azotemia had disappeared (urea 0.27 g/L, creat 9.6 mg/L) and the serum albuminemia was low (20 g/L, UV 22/40; Alb/Glob ratio 0.5). The skin lesions and tachypnea remained unchanged. The cat was still anorexic. Whilst waiting for results, an injection of cefovecin (0.08 mg/kg SC) and dexamethasone (0.2 mg/kg IV) was then administered and the doxycycline treatment was stopped. Clinical recovery was spectacular (immediate recovery of appetite and disappearance of skin lesions Fig. 3). The cat was returned to its owner still following a course of methylprednisolone (4 mg/d PO, 5 days).

Given the results of the leptospirosis PCR, treatment based on doxycycline was restored 14 days after the cefovecin injection (50 mg/d PO) so as to fight against the risk of a chronic carrying state.

Monitoring

A month and a half later, the cat had gained 1.4 kg (BMI 6/9). The treatment based on doxycycline was stopped after 4 weeks. The cat was in a perfect general state (Fig. 4) and there were no longer any visible skin lesions. The heart murmur was still there, but the cat was no longer breathless. Hemoglobulinemia (12.9 g/dL), serum albuminemia (32 g/L) and glycemia (0.88 g/L) were normal as well as the alb/glob ratio (0.97). The Idexx snap FeLV/FIV combo test was negative.

A serology test (MAT technique, Lyons laboratory) was carried out to look for anti-Leptospira antibodies on day 0 and 5 weeks later. This serology analysis showed positive antibodies for Leptospira Sejroë (serotype Saxkoebing) at a rate of 1/400 for the first consultation and at a rate of 1/3200 5 weeks later. This was a clear case of seroconversion. The Ehrlichosis and Anaplasmosis serologies were negative at 5 weeks. Two years after this episode, the cat was still in a good general state.

Discussion

Leptospirosis diagnosis criteria

According to the ACVIM consensus of 2010 [1], the major diagnosis criteria of leptospirosis in dogs are seroconversion and direct evidence of the pathogenic agent's presence. Direct observation of Leptospiras using a traditional optical microscope is very difficult (requires a dark-field microscopic examination). Growing a culture is possible but difficult to put into general practice because these bacteria take from 3 to 6 months to grow [2,14]. Detection of Leptospiras is easier using PCR on blood or urine samples, or by immunohistochemical identification on kidney or liver biopsies. Here we have manifest case of seroconversion (the antibody titer has multiplied by 8 in 5 weeks), and a positive PCR on blood but not on urine, which gives us very strong arguments to conclude that it was a real case of clinical leptospirosis according to the diagnostic criteria of the consensus (Table 1). The infection was probably recent (leptospiuria is expected in 50% of cases in the 7 to 14 days after

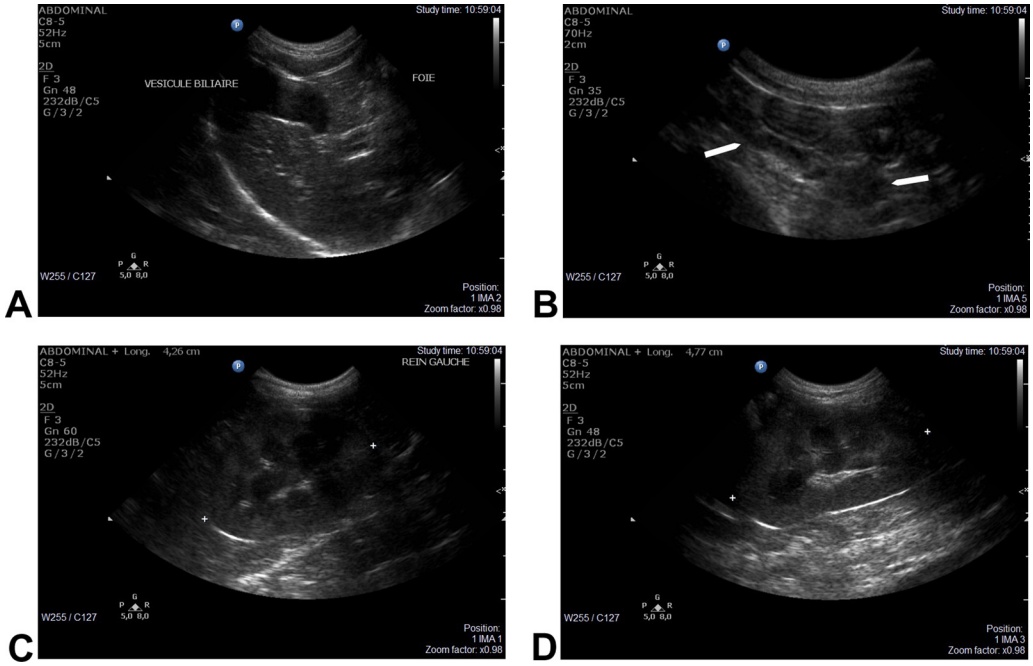

Figure 2. Abdominal ultrasound examination at the first consultation. A. Note the hyperechogenicity of the liver parenchyma (''foie'') and the normal appearance of the gall bladder (''vesicule biliaire''). B. Pancreas (indicated by white arrows) appeared with hypoechogenic center and hyperechogenic perimeter without hyperechogenicity of surrounding fat. C and D. Nephromegaly of the two kidneys was observed without echogenicity modification.

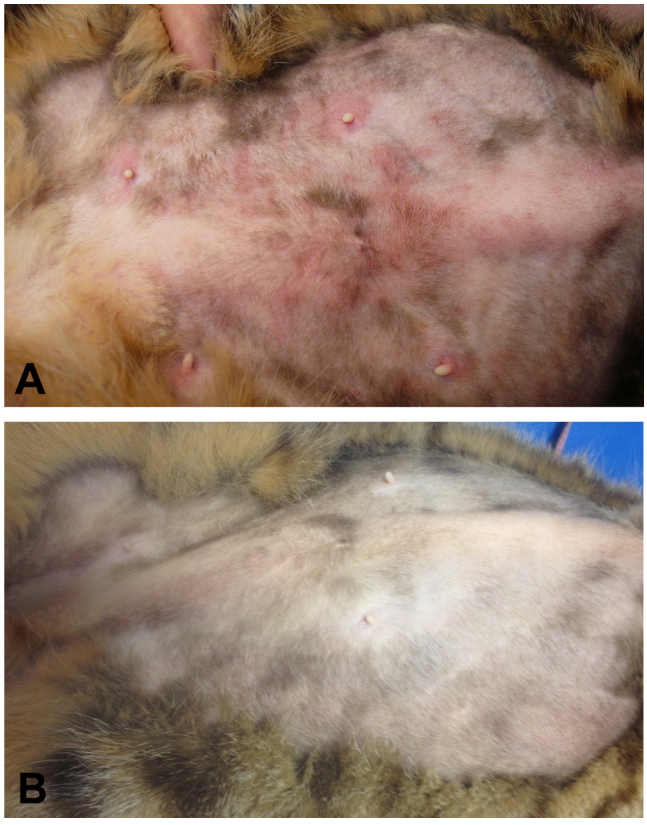

Figure 3. Closer view of abdominal skin lesions before (A) and after (B) corticoid treatment. Hyperemia disappeared in a few hours.

Table 1 Criteria for diagnosis of canine leptospirosis [17] compared to our screening exam results (nt: not tested).

Criteria for diagnosis	In our case
Serologic testing by microscopic agglutination	
Vaccination more than 3 months earlier	Not relevant here
Fourfold rise in paired titers at 3 weeks interval or	8 fold rise in paired titers at 5 weeks interval
800 or greater single titer	Yes: 1/3200
Microscopy	
Lesions compatible by gross or microscopic examination in specimens taken by biopsy or necropsy	nt
Silver or immunostaining of tissues	nt
Organism identification	
Dark-field microscopy of urine	nt
Immunostaining of urine sediment	nt
Polymerase chain reaction positive results on tissues or body fluids	PCR positive result on blood but not in urine
Culture	nt

experimental inoculation) [15,16], and the first antibodies appear after the first week of infection [2,7]. The cat was FeLV and FIV negative (Idexx snap combo test) when controlled at 5 weeks. Immunosuppression by these retroviruses can therefore reasonably be ruled out given the sensitivity of this test, as in the last 3 cases published [13]. Moreover the antibodies directed against *Ehrlichia* spp. or *Anaplasma* spp. were negative 5 weeks after the initial consultation, which proves that we did not have a case of co-infection by these two pathogenic agents.

Correlation with the clinical symptoms of canine leptospirosis

In dogs, leptospirosis can have several clinical forms. A subacute form with kidney and liver failure as well as an acute form with disseminated intravascular coagulation, lung haemorrhages and shock (with a high level of mortality).

Clinical signs associated with the subacute form of canine leptospirosis are listed in Table 2, as described by Greene [17]. The clinical presentation of our cat is comparable to this in some respects. If we attribute points, the clinical examination and the medical history of our cat covers 7 items out of 18. Hyperesthesia is a more specific symptom than anorexia and vomiting in cats. Pain on handling (with allodynia) was probably due to the myalgia described in dogs (a determination of creatine kinase levels could have thrown some light on this aspect). The cat's tachycardia and its murmur were not investigated. In dogs, an increase in cardiotrophin 1 has been described, potentially linked to myocarditis [17]. The cat's tachypnea (restrictive dyspnea) has not been explored by thorax X-ray imaging. It was may be linked to vasculitis or pulmonary hemorrhage, described in cases of clinical leptospirosis in dogs and humans. Pulmonary impairment is moreover a worse prognostic factor in these two species, with a correlation between its intensity and the mortality rate [18]. The pulmonary lesions are partly related in the dog to immune complex deposits [19]. Only the cat's skin lesions are surprising. Vasculitis, due to immune complex is the most likely suspected in view of the clinical presentation and the almost immediate disappearance of the lesions after receiving corticosteroids. Only a skin biopsy, which was not carried out, could have clearly established its origin.

Comparison with previous clinical cases described in cats

The clinical presentation of our case (vasculitis, pain on handling, dehydration and tachypnea without PU/PD, jaundice or fever) is particularly original in comparison to the last 3 clinical cases described in cats [13], where the animals had essentially PU/PD (3 cases) and fever (2 cases) with dehydration (3 cases) without liver disease. Two of these 3 cats had enlarged kidneys, as in our case [13]. Lameness, uveitis, hematuria [13], ascites with impaired liver function, icterus [10], weight loss [2] or widespread haemorrhages [12] have been described as well. Death can occur [2,13]. Skin lesions have never to our knowledge been described in cats suffering from leptospirosis. This particularity is perhaps linked to infection from the Sejroë serogroup (serotype Saxkoebing).

Imaging and blood biochemistry modifications

The cat's blood count modifications were not remarkable. Neutrophilia was not present, unlike 2 of the 3 recent published cases [13]. Only a marked platelet activation, without thrombocytopenia was observed. Coagulation times were not measured. Pre-renal azotemia was expected in the presence of dehydration but the cat did not have kidney failure (creatine levels within range), as in two of the three cases of feline clinical leptospirosis recently described, the last case presenting with kidney failure [13]. Only proteinuria indicated that the kidneys were affected. A urine protein over urine creatinine ratio, electrophoresis of urine proteins and a kidney biopsy would have better characterised the lesions, but these examinations were not carried out. The absence of kidney failure was may be linked to the precocity of the infection and its treatment. In fact, the association of feline kidney failure with Leptospira seropositivity has been recently evoked [6]. Interstitial nephritis directly caused by spirochetes seems to be the most common clinical manifestation of leptospirosis in cats [14]. Our case may have developed kidney failure later on, like the second case described by Arbour et al. [13].

Liver and pancreas enzymes had not been modified. In dogs, pathogenic Leptospira agents have a trophism, which can vary according to serogroups, for kidneys or the liver [20]. Some serogroups in cats have a known liver trophism (Pomona and Australis) [15], unlike other serogroups [2]. The Bratislava serogroup was linked to an invasion of brain and lung capillaries and aqueous humour [12]. It may be that Leptospiras of the Sejroë serogroup in cats do not have a major liver trophism.

If a score is applied to our case (Table 3), the biochemical and hematological analyses come close to cases described for dogs for only 5 items out of 21. On the other hand, the imaging results were compatible, with nephromegaly and an increase in the echogenicity of the renal cortex (2/3). Moreover, the hypoechogenicity of the pancreas (found here) and its increase in size are also non-specific criteria often found in cases of canine leptospirosis [17].

Transmission

Clinical cases described in dogs are more frequent in periods of rainy weather. In the region of Rennes, monthly rainfall was heavier than usual in December 2011 (that is, 5 weeks before, 160 mm) and less marked in January (51 mm, and temperatures of $< 0\,^{\circ}C$, data source: Ouest France) for clinical symptoms beginning at the start of February. The cat lives in close contact with hunting dogs and hunts rodents herself, which appears to be a factor, which promotes contamination [6]. Contamination could have occurred by direct contact with dogs themselves (possible cases of chronic carriers), via rodents (cases of transmission described via bites [21]), or by contact with water polluted with contaminated urine. We do not have any known medical history about recent bite wounds or abscesses. It would have been interesting to know the serological status of the family dogs as regards this serovar, but this was not carried out.

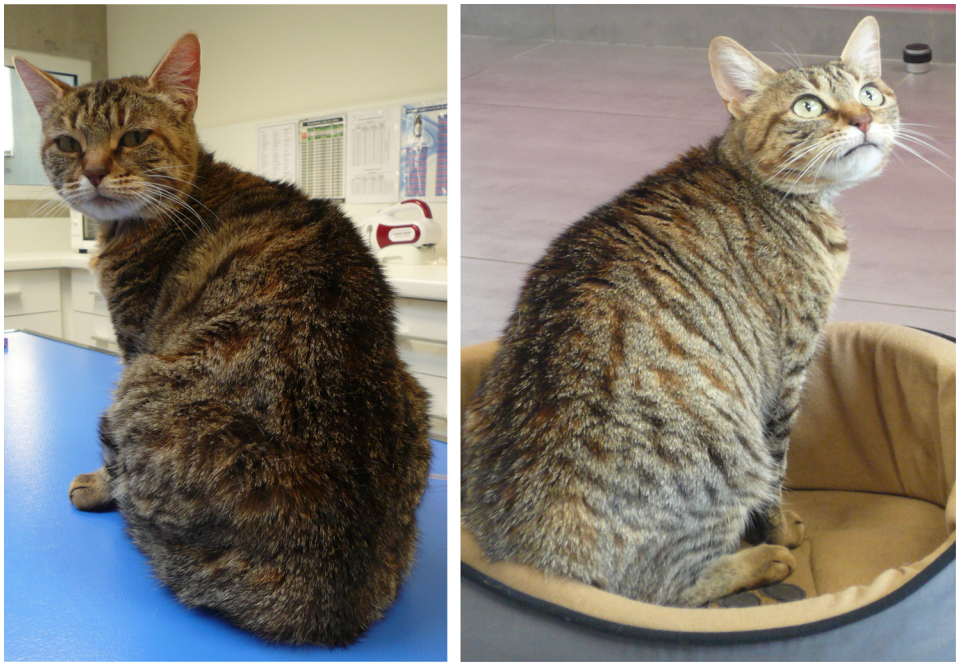

Figure 4. General appearance of the cat at the first consultation: note the dull fur (A). Good general state of the cat at the control 6 weeks later (B).

Table 2 Clinical symptoms of the subacute form of leptospirosis in dogs [17], and comparison with the symptoms presented by our feline case. We compared the symptoms presented by our cat to the subacute symptoms of canine leptospirosis (1: symptom present; 0: symptom not present). The score was 7/18.

Medical problems associated with canine leptospirosis	In our case	Score
History		
Young animals more severely affected than adults	4 years old	0
Large breed, outdoor dogs commonly affected	Recent outdoor access; lives in close contact with hunting dogs; contacts with wild rodents	1
Clinical findings in subacute cases		
Fever	37.6 °C	0
Anorexia	Yes	1
Vomiting	Yes	1
Dehydration	Yes	1
Polydipsia and polyuria	Yes	1
Reluctance to move	No	0
Paraspinal hyperesthesia	Yes	1
Congested mucous membranes	No	0
Petechial or ecchymotic hemorrhages	No, but probable skin vasculitis lesions	
Conjunctivitis	No	0
Uveitis	No	0
Rhinitis	No	0
Tonsillitis	No	0
Oliguria or anuria	No	0
Coughing or dyspnea	Yes (dyspneic restrictive tachypnea)	1
Icterus	No	0

Table 3 Laboratory and imaging findings. We compared the biochemical and imaging modifications noted in the canine subacute form of leptospirosis [17] to the modifications noted in our case (nt: not tested; 1: modification present; 0: modification not present or not tested). The score applied to our case for the biochemical and hematological analyses comes close to cases described for dogs for only 5 items out of 21. The imaging results are comparable, with nephromegaly and an increase in the echogenicity of the renal cortex (score: 2/3).

Laboratory findings in canine subacute form	In our case	Score
Haematology		
Leukocytosis (neutrophilia)	Lymphocytosis	0
Thrombocytopenia	Activated platelets on blood smear +++	0
Prolonged coagulation times	nt	0
Biochemistry		
Variable mineral and electrolyte disturbances ($\downarrow Na^+$, $\downarrow Cl^-$, $\updownarrow K^+$, $\uparrow PO_4$)	nt	0
↑Glu	Yes	1
↑ALT, AST, LDH, ALP activities, ↑serum bilirubin, serum bile acids	No (normal ALT and ALP)	0
↑serum amylase and lipase activities	No: normal spec fPL	0
Azotemia, ↑serum creatinine	Yes	1
↑serum creatinine kinase	nt	0
↑serum C-reactive protein,	nt	0
↑serum cardiac troponin-I	nt	0
↑serum cholesterol	nt	0
↑serum globulin	Yes	1
↓serum albumin	Yes	1
Urinalysis		
Specific gravity ≤ 1.029	No, 1.050	0
Glucosuria	No	0
Tubular or glomerular proteinuria	Yes	1
Bilirubinuria	No	0
↑number of granular casts	No	0
Pyuria	No	0
Hematuria	No	0
Increased urine protein/creatinine ratio	nt	0
Imaging findings		
Interstitial to nodular alveolar densities	nt	0
Ultrasound of urinary system: renomegaly, pyelectasia, ↑cortical echogenicity	Yes	1
	Abnormal pancreatic imaging	1
Electrodiagnostics		
Ventricular tachyarrhythmias	nt	0

Treatment

Treatment implies the prescription of either penicillins or doxycycline, during the acute phase. According to the ACVIM consensus for dogs [1], doxycycline should be the privileged treatment because it can avoid long term carrying of the disease in the kidneys and should be administered at a dose of 5 mg/kg, twice a day for 2 weeks. In this case, we used doxycycline in a single daily dose at a rate of 16 mg/kg/day. Forty-eight hours of doxycycline alone having obtained no clinical improvement, we used corticosteroids and modified the antibiotherapy (cefovecin) still without knowing the nature of the infection. The addition of corticosteroids is debatable, but the clinical benefit of the combination with cefovecin seemed very clear, without a doubt eliminating a generalised inflammation. The use of corticosteroids has been described in dogs in the case of pulmonary lesions [17]. In view of the results of the PCR we returned to the doxycycline based treatment 14 days after the injection of cefovecin so as to avoid the animal becoming a possible chronic carrier and to avoid chronic shedding, as specified by Hartmann et al. [14], for cats, especially for the humans who come into contact with the cat (1.5% of stray cats in Japan have been shown to be chronic carriers of Leptospiras in the kidney [22]).

Prevention of risks of transmission to humans

A case of direct transmission to humans via a contaminated rat bite has been described [23]. In the face of such a case, the possibility of human contamination by animals should not be underestimated, and owners and clinic staff should be warned so as to avoid all direct contact with cat urine and any bite. All the more so when the cat is in pain when being

handled, unlike its usual behaviour. Moreover, it would have been justified to treat the hunting dogs in direct contact with the cat preventively with doxycycline for 2 weeks [1], even if contact with dogs does not seem to statistically increase the risk of Leptospira seropositivity in cats [6].

Conclusion

Feline Leptospira infection should not be underestimated, because many studies have shown serological positivity in cats, with a prevalence of up to 35% in different countries, or even PCR blood or urine positivity. Household cats could be chronic carriers and risk shedding Leptospira in their urine and contaminating human beings. Some cats could even develop clinical leptospirosis and develop subsequent chronic kidney failure. Consequently, when faced with vasculitis, unexplained increased albumin over globulin ratio, kidney failure, PU/PD, uveitis or lameness, feline leptospirosis should be considered in the list of differential diagnoses, in particularly in outdoor cats that hunt. Moreover, the Sejroë serogroup should be included in serological testing, with paired titer tests at a 3-week interval.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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