Hemotropic mycoplasmas in cats
Part 2: case report

Hemotrofe mycoplasmen bij katten
Deel 2: casuïstiek

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ABSTRACT

An eight-month-old apathic cat was referred to the Department of Medicine and Clinical Biology of Small Animals of the Ghent University Faculty of Veterinary Medicine, Merelbeke, Belgium. The cat had a severe case of non-regenerative anemia with a hematocrit of only 2.9%. Cytological examination of a bone marrow aspirate led to the diagnosis of pure red cell aplasia (PRCA). Additionally, a PCR assay for "*Candidatus* Mycoplasma haemominutum" ("*Candidatus* M. haemominutum") DNA was positive. Although unproven, an infection with "*Candidatus* M. haemominutum" could have contributed to the immune-mediated destruction of red blood cell precursors. The cat recovered completely after treatment, which consisted of multiple blood transfusions, antimicrobial agents, and long-term prednisolone therapy (10 months). There were no signs of clinical relapse at 20 months after cessation of therapy.

SAMENVATTING

Een acht maanden oude kat werd in apatische toestand doorverwezen naar de vakgroep Geneeskunde en Klinische Biologie van de Kleine Huisdieren van de Faculteit Diergeneeskunde in Merelbeke, België. De kat had een erge niet-regeneratieve anemie met een hematocriet van slechts 2,9%. Het cytologisch onderzoek van een beenmergaspiraat leidde tot de diagnose van "pure red cell aplasia" (PRCA). Daarnaast was een PCR (polymerase chain reaction)-test voor "*Candidatus* Mycoplasma haemominutum" DNA positief. Hoewel niet bewezen, zou de infectie met "*Candidatus* M. haemominutum" kunnen bijgedragen hebben tot een immuungemedieerde vernietiging van de rode bloedcelprecursoren. Na een langdurige prednisolonebehandeling (10 maanden), meerdere bloedtransfusies en een antimicrobiële therapie was de kat volledig hersteld. Twintig maanden na de stopzetting van de therapie waren er nog steeds geen tekenen van klinisch herval.

INTRODUCTION

Feline hemotropic mycoplasmosis (FHM) is the new name given to the disease formerly known as feline infectious anemia or hemobartonellosis (Sykes, 2003). The causative agents of this disease are hemotropic mycoplasmas (hemoplasmas). So far, three feline hemoplasma species have been described: *Mycoplasma haemofelis* (*M. haemofelis*), "*Candidatus* Mycoplasma haemominutum" ("*Candidatus* M. haemominutum") and "*Candidatus* Mycoplasma turicensis" ("*Candidatus* M. turicensis") (Neimark *et al.*, 2001, 2002; Foley and Pedersen, 2001; Willi *et al.*, 2005).

This case report describes a young male cat with pure red cell aplasia. An acute infection with "*Candidatus* M. haemominutum" could have contributed to an immune-mediated pure red cell aplasia.

CASE REPORT

A five-month-old intact male *Felis vulgaris* was brought to the referring veterinarian, with lethargy being the main complaint. His mucous membranes (mouth and eyes) were very pale. The cat was mostly kept indoors, together with another healthy cat, and was regularly vaccinated (Purevax RCPCh®, Merial) and dewormed (Catminth®, Pfizer Animal Health).

Blood analysis was performed and included complete blood count (CBC), biochemistry and viral serology. The results revealed severe microcytic, normochromic anemia and mild lymphocytosis. The serological tests for Feline Leukemia Virus (FeLV), Feline Immunodeficiency Virus (FIV) and Feline Coronavirus were negative. A detailed overview of the blood test results is presented in Table 1. The cat was treated with prednisolone injections 6.25 mg/kg (Prednisolone® 2.5%, Kela, Hoogstraten, Belgium) SC, SID, over a period of seven days. Thereafter, five days of methylprednisolone 1 mg/kg (Moderin® 2 mg, Pfizer Animal Health, Louvain-la-Neuve, Belgium) PO, SID was given. This led to clinical improvement and the pale mucous membranes turned pink again. Two months later, the cat gradually became lethargic again and 0.5 mg/kg methylprednisolone (Moderin® 2 mg, Pfizer Animal Health) was administered PO every other day. Its clinical condition slowly deteriorated: the lethargy worsened, the mucous membranes became pale again and the cat developed anorexia. A blood analysis at that time revealed a more severe microcytic anemia. The reticulocyte percentage was mildly increased. Furthermore, there were increased serum iron and serum urea concentrations, as well as increased liver enzyme activity (aspartate aminotransferase (AST) and alanine aminotransferase (ALT)) and hypokalemia (Table 1). Microscopic examination of a blood smear showed no signs of infection with hemoplasmas, *Babesia* species or heartworm (*Dirofilaria immitis*). On the same day, the cat was referred to the Department of Medicine and Clinical Biology of Small Animals of the Ghent University Faculty of Veterinary Medicine, Merelbeke, Belgium for further examination and treatment. Physical examination showed that the animal was apathic and in poor nutritional condition (2.150 kg bodyweight). The cat was hypothermic (rectal temperature 35.0° C), the mucous membranes were very pale, and the capillary refill time could not be determined. There were no other remarkable findings on physical examination. CBC, serum sodium and potassium concentrations (Table 2), as well as the SNAP® Combo Plus FIV antibody/FeLV antigen test kit, (IDEXX Laboratories, Maine, USA) and a blood smear were all repeated (Table 2). Additionally, a high specificity real-time PCR assay (Scanelis, Toulouse, France) for *Mycoplasma haemofelis* and "*Candidatus* Mycoplasma haemominutum" was performed. The results of the SNAP® test were negative. On the blood smear, platelet aggregates were visible and there was anisocytosis and polychromasia of the erythrocytes. Autoagglutination was negative and a direct Coombs test could not be performed due to an insufficient blood sample.

After blood typing (type A blood), a transfusion of fresh whole blood was started, followed by an intravenous infusion of NaCl 0.9%, and the cat was warmed with an infrared light. After the transfusion, the hematocrit increased to 10% (reference range 25-45%) and the clinical condition improved. Subsequently, thoracic radiographs and abdominal ultrasonography

Table 1. Results of the first two blood tests.

	02 Oct 2006	08 Jan 2007	Reference ranges	Units
Hematology				
Hemoglobin	**1.56**	**0.75**	4.98-9.34	mmol/L
Hematocrit	**6.9**	**2.9**	24.0-45.0	%
Erythrocytes	**1.94**	**1.08**	5.00-10.00	10^{12}/L
MCV	**35.8**	**26.5**	37.0-55.0	fL
MCH	12.8	**11.5**	12.0-18.0	pg
MCHC	35.7	**43.2**	30.0-36.0	g/dL
Leukocytes	14630	8030	5000-15000	/µL
Total immature neutrophils	0	0	< 550	10^6/L
Total segmented neutrophils	7096	4958	3600-10500	10^6/L
Total lymphocytes	**5647**	2987	900-4200	10^6/L
Total monocytes	410	137	< 550	10^6/L
Total basophils	15	0	0-80	10^6/L
Total eosinophils	**1448**	32	< 800	10^6/L
Platelets	458000	320000	175000-500000	/µL
Reticulocytes	nd	**2.1**	< 1.3	%
Iron	nd	**278**	110-170	µg/dL
Biochemistry				
Sodium	nd	156	145-158	mmol/L
Potassium	nd	**2.6**	3.0-5.0	mmol/L
Urea	**12.32**	**26.97**	6.66-11.65	mmol/L
Creatinine	69	87.5	8.8-132.6	µmol/L
Total proteins	65	74	56-78	g/L
Albumin	34.8	43.7	25.0-45.0	g/L
Total bilirubin	0.10	< 0.10	< 0.40	mg/dL
AST	33	**170**	< 46	U/L
ALT	**57**	**214**	< 43	U/L
Gamma-glutamyltransferase	< 3	< 3	< 9	U/L

nd= not determined

Table 2. Results of the blood analyses performed during hospitalization (08 Jan 2007 (day 1 of hospitalization) until 16 Jan 2007 (day 9 of hospitalization)).

	08 Jan day 1	09 Jan day 2	10 Jan day 3	11 Jan day 4	12 Jan day 5	13 Jan day 6	14 Jan day 7	16 Jan day 9	Reference ranges	Units
Hematology										
Leukocytes	7.1	**5.5**	6.1	7.3	7.6	7.3	10.2	**5.6**	6.0-11.0	10^9/L
Erythrocytes	**1.08**	**3.55**	**2.40**	**2.19**	**2.27**	**1.93**	**1.85**	8.78	5.00-10.00	10^{12}/L
Hemoglobin	**0.0**	**3.2**	**2.4**	**2.1**	**2.1**	**1.8**	**1.8**	6.7	5.0-9.3	mmol/L
Hematocrit	**4.0**	**16.0**	**10.8**	**9.8**	**10.3**	**8.9**	**8.7**	36.8	25.0-45.0	%
Platelets	278	477	271	246	198	205	195	749	180-550	10^9/L
MCV	**37**	45	45	45	45	46	47	42	40-55	fL
MCH	**0.00**	0.91	0.98	0.96	0.94	0.94	0.96	**0.76**	0.81-1.05	fmol
MCHC	**0.0**	20.2	21.9	21.3	20.7	20.3	20.3	**18.1**	19.2-22.3	mmol/L
# lymphocytes	2.3	2.2	2.9	3.0	2.8	**3.3**	**4.1**	1.6	1.2-3.2	10^9/L
# monocytes	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.1**	**0.0**	0.3-0.8	10^9/L
# granulocytes	4.8	3.3	3.2	4.3	4.8	4.0	6.0	4.0	1.2-6.8	10^9/L
Biochemistry										
Sodium	**156.8**	nd	**156.4**	150	145.9	154.1	155.7	nd	143-156	mmol/L
Potassium	**2.35**	nd	**3.11**	**2.7**	**2.91**	**3.15**	3.70	nd	3.5-5.2	mmol/L

nd = not determined

were performed to identify underlying causes. The thoracic radiographs showed signs of left-sided heart dilatation, and abdominal ultrasonography showed a liver with homogeneous parenchyma displaying the presence of congestive vasculature. Echocardiography did not reveal any structural abnormalities of the heart.

The most important differentials at this time were infectious causes, immune-mediated causes or a primary bone marrow disorder. Therefore, doxycycline 10 mg/kg (Ronaxan® 20, Merial Belgium, Toulouse, France) PO, SID (together with food) and prednisolone 1 mg/kg (Prednisolone® 5 mg, Kela, Hoogstraten, Belgium) PO, BID were prescribed. Further, ranitidine 2 mg/kg (Zantac®, GlaxoSmithKline, Genval, Belgium) IV, BID, was prescribed. The CBC and serum sodium and potassium concentrations were followed daily (Table 2).

On the 4th day of hospitalization, the NaCl infusion was removed because the cat started to eat independently, and as from then ranitidine was given PO (Zantac® syrup, GlaxoSmithKline). The prednisolone dose was increased to 1.75 mg/kg PO, BID. Potassium gluconate 2 mEq K^+ (Ultra-K® syrup, 20 mEq K^+/15 ml, Melisana, Turnhout, Belgium), PO, BID was supplemented because of hypokalemia. The result of the PCR test for hemoplasmas was available on day 5 and the cat was found to be positive for "*Candidatus* M. haemominutum" DNA. On the 8th day of hospitalization, a second transfusion of fresh whole blood was administered and a bone marrow aspiration was performed under superficial general anesthesia (buprenorphine 0.01 mg/kg IV (Temgesic®, Schering-Plough, Hull, United Kingdom), propofol 2 mg/kg IV (Rapinovet®, Schering-Plough, Wicklow, Ireland). On cytological examination of the aspirate, various megakaryocytes were visible. There was erythrocytic aplasia (myeloid-to-erythroid ratio > 75:1) and a mild to moderate granulocytic hypoplasia. Based on the bone marrow specimen, pure red cell aplasia (PRCA) was diagnosed. The bone marrow aspirate was tested with a PCR assay for FeLV, but the result was negative. On day 9, the hematocrit had returned to reference ranges and the cat was sent home. The following treatment was continued at home: ranitidine 2 mg/kg (Zantac® syrup) PO, BID, 30 minutes before the meal, doxycycline 10 mg/kg (Ronaxan® 20) PO, SID (together with food) and prednisolone 1.75 mg/kg (Prednisolone® 5 mg) PO, BID.

Two weeks later, the cat was alert and had gained weight (bodyweight 2.25 kg). The hematocrit at that time was 20.3%, with a normal reticulocyte percentage (1.1%; reference range <1.3%). There was a leukopenia (2720 /µL; reference range 5000-15000 /µL) with a decrease in the number of segmented neutrophils (1707 x 10^6/L; reference 3600-10500 x 10^6/L). Treatment with ranitidine and doxycycline was discontinued, but prednisolone 1.7 mg/kg (Prednisolone® 5 mg) PO, BID, was further administered. A second bone marrow punction was recommended if the leukopenia would persist.

The dosage of prednisolone was slowly tapered over the succesive 10 months and was then discontinued. Twenty months after diagnosis, the cat is in good health and the results of CBCs, performed on a regular basis, are within reference ranges.

DISCUSSION

The cat in this report presented with severe microcytic anemia. The reticulocyte percentage was mildly increased, but the regeneration was highly insufficient in relation to the extremely low hematocrit. The cat was therefore diagnosed with a non-regenerative anemia. The major differential diagnosis of non-regenerative anemia in cats are bone marrow disorders, infectious diseases, anemia due to chronic disease,

anemia due to renal disease, acute blood loss (first 48-96 hours) or anemia releated to endocrine disorders (Couto, 2003). Anemia due to chronic or endocrine disease almost never results in hematocrit below 17 or 18% (Couto, 2003) and the history and the results of blood analysis did not support renal or endocrine disorder or acute blood loss.

Neoplastic, hypoplastic or dysplastic bone marrow disorders can result in anemia and/or other cytopenias. To determine a final diagnosis, histopathological or cytological examination of a bone marrow specimen are warranted (Couto, 2003). In this case, cytology of a bone marrow aspirate was performed and PRCA was diagnosed.Various causes for bone marrow aplasia or hypoplasia are described in cats, e.g. FeLV, immune-mediated, drug-induced or idiopathic (Couto, 2003). Cats with PRCA usually have a hematocrit below 15% and CBC usually shows a severe (normocytic, normochromic) non-regenerative anaemia (Couto, 2003). Pure red cell aplasia is a form of primary immune-mediated hemolytic anemia in dogs and cats that is characterized by erythroid aplasia in the bone marrow (Weiss, 2008). In the present case, the SNAP® Combo FIV antibody/FeLV antigen test and the PCR assay for FeLV were performed on serum and on bone marrow aspirate, respectively. Both test results were negative. No drugs were administered that could have led to bone marrow degradation. In this case, an immune-mediated or an idiopathic condition is most likely. PRCA with a suspected immune-mediated cause is relatively common in cats (Couto, 2003; Weiss, 2008). The pathogenesis would be similar to that of immune-mediated hemolytic anemia: the present antibodies (or cell-mediated immunity) are directed against the erythroid precursors (Couto, 2003).

The PCR analysis of the blood was positive for "*Candidatus* M. haemominutum" DNA. This positive result could be due to an acute or a chronic infection (carrier state). If the cat had just carried "*Candidatus* M. haemominutum", then this agent did not cause the clinical signs. In that case, the PRCA would be idiopathic, because no other immune-mediated cause of the PRCA could be found. An acute infection with "*Candidatus* M. haemominutum" contributing to an immune-mediated destruction of the red blood cell precursors cannot be excluded. PCR assays for *Cytauxzoon felis*, *Ehrlichia* species or *Anaplasma phagocytophilum* were not performed because these are rare infectious causes of anemia in cats, the risk of exposure to them is geographically defined (Ishak *et al.*, 2007), and they have not been reported in Belgium.

Quantitative real-time PCR assays allow quantification of hemoplasma DNA, which allows for an estimation of the clinical significance of an infection (Willi *et al.*, 2007b). Although the real-time PCR assay performed in this case was not a quantitative one, the laboratory estimated on the basis of its experience that in this sample the bacterial load was relatively high (estimation: 100,000 bacteria/ml blood) and thus most likely clinically relevant (Jean-Luc Pingret, Scanelis, personal communication). This makes an acute infection with "*Candidatus* M. haemominutum" likely.

In 2003, Sykes reported that an acute infection with "*Candidatus* M. haemominutum" in immunocompetent cats usually results in mild or absent clinical signs. Willi *et al*. (2007b) added that an acute infection with "*Candidatus* M. haemominutum" usually does not lead to a significant decline of the hematocrit. Therefore, it is questionable whether the acute infection with "*Candidatus* M. haemominutum" was actually the cause of the disease in this case. On the other hand, a recent study showed that cats experimentally infected with "*Candidatus* M. haemominutum" showed a significant decline in hematocrit, which could indicate the destruction of erythrocytes (Tasker *et al*., 2006). Coinfection of "*Candidatus* M. haemominutum" and "*Candidatus* M. turicensis" can result in a significantly lower hematocrit compared to hemoplasma PCR-negative cats (Willi *et al*., 2006b). In this patient, the blood sample was not tested for the presence of "*Candidatus* M. turicensis" DNA.

Cats are considered fully immunocompetent from the age of 12 months. There are still substantial changes in the immune system during the first year of life (Day, 2007). The cat in this report became infected at a young age, making an acute infection with "*Candidatus* M. haemominutum" a possible cause of illness.

Clinical feline hemotropic mycoplasmosis is mainly characterized by extravascular hemolysis (Willi *et al*., 2007b). The most common hematologic abnormalitiy is regenerative, macrocytic and normochromic anemia (Willi *et al*., 2007b). Here, the cat had a non-regenerative microcytic, normochromic anemia. During hospitalization and treatment, the anemia became normocytic. Microcytic, hypochromic anemia is typically seen together with iron deficiency (Couto, 2003). In this case, the serum iron concentration was high, a fact which was likely related to hemolysis (Andrews and Smith, 2000). Although feline hemotropic mycoplasmosis usually results in regenerative anemia, some infected cats show only moderate regeneration, for instance due to underlying retroviral infections (Willi *et al*., 2007b). The patient tested negative for FIV and FeLV. However, non-regenerative anemia has already been described in FeLV-negative cats infected with feline hemotropic mycoplasmosis (Sykes, 2003).

Doxycycline 5-10 mg/kg PO q12-24h for 14 to 21 days is recommended for treating infections with "*Candidatus* M. haemominutum". FeLV-negative cats with PRCA usually respond well to immunosuppressive doses of corticosteroids (4 to 8 mg/kg prednisone PO, SID). This can be combined with chlorambucil (Leukeran®) 20 mg/m^2 PO every two weeks. Responses occur in 70 to 80% of the patients, but recovery may take two to three months andlifelong treatment is usually necessary (Couto, 2003). The cat in this case was treated with doxycycline 10 mg/kg (Ronaxan® 20) PO, SID for 24 days and prednisolone 1.75 mg/kg (Prednisolone® 5 mg) PO, BID for ten months, and it responded well. It is important to note that, after an acute infection with hemoplasmas, cats can remain

chronic carriers for months to years, even after treatment with appropriate antimicrobial agents. A carrier state after infection with "*Candidatus* M. haemominutum" is frequently encountered. A PCR assay to evaluate carrier status in this cat was not performed. Healthy carriers constitute a source of infection for other cats and reoccurance has been described (Willi *et al*., 2007b).

Currently, still little is known about the routes of transmission of hemoplasmas. Blood-sucking arthropods like fleas and ticks probably play an important role in the indirect transmission of the hemoplasmas (Messick, 2003; Willi *et al.*, 2006a). Furthermore, a direct transmission of hemoplasmas between cats has also been suggested. Therefore, special emphasis should be placed on the control of ectoparasites in the prevention of this disease. (Willi *et al.*, 2007a; Sykes *et al.*, 2008).

CONCLUSION

The PRCA in this cat was of an immune-mediated or idiopathic origin. Primary immune-mediated anemia may be more common in cats than previously recognized (Kohn *et al.*, 2006; Weiss, 2007). The infection with "*Candidatus* M. haemominutum" was most likely acute and could have contributed to the clinical signs.

ACKNOWLEGDEMENTS

The authors would like to thank Dr. E. Vandersmissen for the referral of the cat and Dr. J.L. Pingret, Scanelis, France, for his help with the interpretation of the results of the real-time PCR assay.

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