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CASE REPORT

Erosive polyarthritis associated with *Mycoplasma gateae* in a cat

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Erosive polyarthritis was diagnosed in an 11-month-old neutered male Egyptian Mau-cross cat with concurrent glucocorticoid-responsive dermatitis. Clinical signs, synovial fluid analysis, serological tests and radiographic appearance could not differentiate between immune-mediated and infective arthritis. *Mycoplasma gateae* was isolated by strictly anaerobic culture of the synovial fluid. Treatment with Enrofloxacin led to a rapid improvement of the cat's condition. Two months later the cat was euthanased because of severe glomerulonephritis and direct Coombs' test positive anaemia, possibly caused by mycoplasma infection. *M gateae* could not be isolated at post-mortem examination.

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Polyarthritis, causing clinical signs and involving two or more joints is uncommon in cats (Carro 1994). If no infectious agent can be identified, immune-based arthritis (feline non-infective polyarthritis) is suspected. According to radiographic appearance immune-based polyarthritis is divided into erosive and non-erosive forms (Bennett and Nash 1988). This differentiation can be very difficult particularly in the early stages, as radiographic signs appear later in the disease course (Carr and Michels 1997). The main types of erosive immune-based polyarthritis are the periosteal proliferative form with marked periosteal new bone formation

and the rheumatoid form, resembling rheumatoid arthritis in humans and dogs. Non-erosive arthritis is seen in cases of systemic lupus erythematosus (uncommon), in association with a chronic infective process elsewhere in the body, gastrointestinal disease and neoplastic diseases (Bennett and Nash 1988).

Infective polyarthritis (erosive and non-erosive forms) can be caused by a variety of agents including bacteria, L-form bacteria, mycoplasmas and calicivirus (Abercromby 1994). Although mycoplasma-induced polyarthritis is always listed as a cause of feline infective polyarthritis, only three cases have been documented: one case was caused by *M gateae* (Moise et al 1982), one by an unspecified isolate (Pederson 1983) and one by *Mycoplasma felis* (Hooper et al 1985).

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The polyarthritis caused by *M. gateae* was experimentally reproduced in six specific pathogen-free kittens by intravenous injection (Moise et al 1982).

An 11-month-old neutered male Egyptian Mau-cross cat was admitted to the University of Veterinary Medicine, Vienna, because of chronic, pruritic skin lesions localised on the head and neck. The skin problem had started 4 months earlier and transiently responded to glucocorticoid treatment (dose unknown). The cat had been tested negative for feline coronavirus (FCV, immunochromatography), feline leukaemia virus (FLV, enzyme-linked immunosorbent assay), and feline immunodeficiency virus (FIV, immunochromatography).

A miliary dermatitis limited to the neck and head with bacterial infection and secondary excoriations was diagnosed (cytology: eosinophils +++, neutrophils +++, macrophages ++, bacteria ++). Skin scrapings were negative and neither fleas nor flea faeces were found. Changes in blood count (normocytic, normochromic, non-regenerative anaemia, plasma not icteric or haemolytic, reticulocytes low, see Table 1) were interpreted as a consequence of the chronic inflammatory skin disease. The cat was treated with local chlorhexidine, cephadroxil monohydrate (Cefazid; Aristavet, 20 mg/kg bid) and prednisolone (Prednisolon Agepha; Agepha, 0.5 mg/kg bid). A hypoallergenic trial diet was started.

The skin lesions improved and had healed completely within 1 week of treatment.

During this time the cat suddenly became lethargic, pyrexia (40.3°C) and was unwilling to ambulate. All palpable joints, especially the hocks and the carpi were hot and painful with joint capsule distension and periarticular soft-tissue swelling. The mandibular and popliteal lymph nodes were about twice their normal size. Microscopic examination of the fine needle aspirate of a popliteal lymph node revealed reactive hyperplasia. The haematocrit was 22% (0.22 l/l) and the white blood count showed absolute neutropenia (see Table 1). The results of a blood chemistry panel (glucose, creatinine, urea, total protein, albumin, alanine aminotransferase, phosphorus) were within reference limits. Urine specific gravity was 1.047 and a urine dipstick analysis was without abnormalities.

Radiographs of the carpi and the hocks showed periarticular soft tissue swelling and widening of the joint spaces secondary to joint effusion. Bones adjacent to the tarsal joints showed decreased radiodensity and a coarse trabecular

pattern. Signs of focal subchondral bone lysis were seen in the distal epiphysis of the tibia and fibula, talus, calcaneus, tarsal bones, and proximal metatarsi (Fig 1).

Arthrocentesis of one carpal and one tarsal joint was performed under general anaesthesia. The synovia was turbid, of watery consistency and had a total protein content of 5.2 g/dl (52 g/l) (refractometry, normal < 2.5 g/dl (25 g/l)). A direct smear was prepared and the cell number was estimated by counting the number of cells per high-power field (395 cells/high-power field/400×; normal < 2 cells/field, Jacques et al 2002; 86.5% neutrophils and 13.5% mononuclear cells – predominantly macrophages, some rago-cytes/macrophages with multiple small purple cytoplasmic inclusions, thought to represent nuclear remnants or phagocytosed immune complexes). The diagnosis at that time was inflammatory joint disease with reactive hyperplasia of the lymph nodes. Differential diagnosis included immune-based erosive arthritis (rheumatoid arthritis or an early form of feline periosteal proliferative polyarthritis) and infective polyarthritis. The cat tested negative for anti-nuclear antibodies (titre < 1:40, anti-cat IgG (H + L); Hep-2-cell slide, The Binding Site) rheumatoid factor (modified Rose – Waaler test), FIV (antibodies – immunochromatography, antigen in synovia – real time polymerase chain reaction), FCV (antibodies – immunochromatography) and FLV (Antigen Test Kit; IDEXX) infections. As a test for feline rheumatoid factor was not available, a human test kit (Cellognost; RF, Dade Behring) was used. A false negative result is, therefore, possible.

The antibiotic was changed to erythromycin (Erystad; Stada, 20 mg/kg tid). Three days later the cat's condition had not improved and icteric mucous membranes were noticed (haematocrit 21.2% (0.21 l/l), normal > 27% (>0.27 l/l); bilirubin 4.29 mg/dl (73.4 µmol/l), normal < 0.2 mg/dl (<3.4 µmol/l); liver enzymes normal). The haematocrit dropped to 12.9% (0.13 l/l) within 2 days, accompanied by leukocytosis with a left shift (see Table 1). Haemolytic anaemia was suspected and erythromycin stopped. There was no spontaneous agglutination of red blood cells, and blood parasites were not seen on microscopic examinations. Bacteriological examination of synovial fluid from both joints revealed abundant growths of mycoplasmas on modified Hayflicks' medium only under anaerobic conditions (Spergser et al 2002). Mycoplasma isolates were identified as *M. gateae* by colony immunoblotting,

Table 1. Results of blood tests

Day/month/year	29.06.05	01.07.05	05.07.05	08.07.05	11.07.05	19.07.05	01.09.05	Reference range
Erythrocytes $\times 10^6/\mu\text{l}$ ($\times 10^{12}/\text{l}$)	4.69 (4.69)	5.15 (5.15)	na	4.96 (4.96)	3.34 (3.34)	4.85 (4.85)	1.09 (1.09)	5.5–10 (5.5 – 10)
Haemoglobin g/dl (g/l)	6.5 (65)	7.3 (73)	na	6.7 (67)	4.6 (46)	6.5 (65)	1.1 (11)	8–14 (80–140)
Haematocrit % (volume fraction)	20.4 (0.20)	21 (0.21)	22 (0.22)	21.2 (0.21)	12.9 (0.13)	18.9 (0.19)	6.2 (0.06)	27 – 47 (0.27–0.47)
Mean corpuscular volume μ^3 (fl)	43.5 (43.5)	40.8 (40.8)	na	42.7 (42.7)	38.6 (38.6)	39 (39)	56.9 (56.9)	40–55 (40–55)
Mean corpuscular haemoglobin pg/cell (pg/cell)	13.9 (13.9)	14.2 (14.2)	na	13.5 (13.5)	13.8 (13.8)	13.4 (13.4)	15.6 (15.6)	13–17 (13–17)
Mean corpuscular haemoglobin concentration g/dl (g/l)	31.9 (319)	34.8 (348)	na	31.6 (316)	35.7 (357)	34.4 (344)	27.4 (274)	31–34 (310 –340)
Reticulocytes/ μl ($\times 10^9/\text{l}$)	10 318 (10.32)	na	na	68 448 (68.45)	29 392 (29.39)	34 920 (34.92)	39 567 (39.57)	>60 000 (>60)
Reticulocyte index	0.06	na	na	0.4	0.13	0.19	0.26	>2 in regenerative anaemia
Leukocytes/ μl ($\times 10^9/\text{l}$)	4120 (4.12)	12 290 (12.29)	3620 (3.62)	42 730 (42.73)	62 420 (62.42)	16 340 (16.34)	18 240 (18.24)	6000–18 000 (6–18)
Neutrophils band./ μl ($\times 10^9/\text{l}$)	0.04 (0.00)	0.12 (0.00)	217.2 (0.22)	3845.7 (3.85)	0.62 (0.00)	0.16 (0.00)	0,18 (0.00)	<500 (<0.5)
Neutrophils segm./ μl ($\times 10^9/\text{l}$)	2142.49 (2.14)	7877.89 (7.88)	832.6 (0,83)	32 474.8 (32.48)	58 986.9 (58.99)	13 889 (13.89)	14 427 (14.43)	3600–12 750 (3.6–12.75)
Lymphocytes/ μl ($\times 10^9/\text{l}$)	1648 (1.65)	3687 (3.69)	2027.2 (2.03)	5127.6 (5.13)	1685 (1.69)	1405.24 (1.41)	2225 (2.23)	900–5100 (0.9–5.1)
Monocytes/ μl ($\times 10^9/\text{l}$)	41.2 (0.04)	430.15 (0.43)	362 (0.36)	854.6 (0.86)	1185 (1.19)	702.62 (0.7)	784.32 (0.78)	<500 (<0.5)
Eosinophils/ μl ($\times 10^9/\text{l}$)	288.4 (0.29)	221.22 (0.22)	181 (0.18)	427.3 (0.43)	249.68 (0.25)	310.46 (0.31)	273.6 (0.27)	<800 (<0.8)
Thrombocytes $\times 10^3/\mu\text{l}$ ($\times 10^9/\text{l}$)	na	na	na	na	184 (184)	275 (275)	136 (136)	180–430 (180–430)

na = Not applicable.



Fig 1. Radiographs of the left hock in a medio-lateral and dorso-plantar view. Soft-tissue swelling is visible; there are radiolucent focal areas representing erosive lesions of the bone (arrows).

using specific rabbit hyperimmune sera against feline mycoplasma species (Rosengarten and Yogev 1996). Enrofloxacin (Baytril; Bayer, 5 mg/kg sid, prednisolone was stopped) was administered, leading to a dramatic improvement of clinical signs including lameness, after 1 day of treatment.

One week later the cat had responded well to treatment. Joints and lymph nodes were almost of normal size. The haematocrit had increased to 18.9% (0.19 l/l) and neutrophils and monocytes were only mildly elevated (see Table 1). Liver enzymes and creatinine were within the reference range.

Two weeks after stopping antibiotic treatment (duration of treatment 16 days) the cat relapsed (fever 40.3°C and swollen painful joints), but rapidly improved again by reintroducing Enrofloxacin therapy.

Enrofloxacin was stopped after 11 days of treatment (against our advice). Shortly thereafter the cat became lethargic and anorexic. Prednisolone therapy was started (0.8 mg/kg once daily, with no veterinary consultation) without clinical improvement.

When the cat was presented at our clinic it was lethargic, recumbent, and severely dehydrated. Body temperature was 35.7°C. The joints were normal (not swollen or painful), but the lymph nodes were again about twice their normal size. The

direct antiglobulin test (direct Coombs' test, Feline Antiglobulin Test, ICN Biomedicals, Inc) was positive (>1:64) at 4°C and 37°C. Severe non-regenerative autoimmune haemolytic anaemia (Table 1) was diagnosed. Further examinations revealed mild thrombocytopenia (no aggregates), leukocytosis, hypoproteinaemia (albumin and globulin), abnormal liver enzyme assays, and azotaemia (creatinine 4.3 mg/dl (380 µmol/l), normal < 1.6 mg/dl (<141 µmol/l); urea 481 mg/dl (171 mmol/l), normal < 65 mg/dl (<23 mmol/l); phosphorus 17.5 mg/dl (5.65 mmol/l), normal < 10.2 mg/dl (<3.29 mmol/l) in cats < 1 year). The specific gravity of the urine (cystocentesis) was 1.014. An abdominal ultrasonographic examination showed enlarged hyperechoic kidneys (left kidney 4.01 × 2.37 cm, right kidney 4.1201 × 2.35 cm). With a protein/creatinine ratio of 9.1 (normal < 1, Hitachi 911, Roche) and the presence of granular and hyaline casts with very few leukocytes and no erythrocytes in the urine, glomerulonephritis was suspected. Radiographs of the hocks and carpi showed no periarticular soft-tissue swelling. Bones of the tarsal joints had decreased bone radiodensity with hardly visible erosive lesions.

The combination of non-regenerative anaemia, autoantibodies, and kidney failure led to the decision to euthanase the animal. *M. gateae* could not be isolated from the pharynx, mandibular lymph

nodes, lung, urine, cerebrospinal fluid or joints at post-mortem examination. Autopsy confirmed a severe chronic nephritis in terms of focal hydropic tubular degeneration, focal interstitial plasmacytic nephritis, membranoproliferative glomerulonephritis, multiple atrophic glomerulae, and fibrosis. The lymph nodes showed reactive hyperplasia. Focal subtle fascicular fibrillation could be demonstrated on the surface of the articular cartilages, and discreet villous hyperplasia without inflammatory infiltration was found within the synovial membrane.

Feline mycoplasmas are normal inhabitants of conjunctival membranes, upper respiratory tract, and urogenital tract in cats (Carter and Wise 2004) and were isolated in approximately 80% (Blackmore et al 1971), 69% (Tan et al 1977) and 35% (Randolph et al 1993) of healthy cats. They are not usually recovered from the lower airways of healthy cats (Randolph et al 1993), where they have been associated with pyothorax (Malik et al 1991), pneumonia and abscessation (Foster et al 2004). Mycoplasma species have also been implicated as aetiological agents in feline conjunctivitis (Haesebrouck et al 1990), ulcerative keratitis (Gray et al 2005), polyarthritis (Moise et al 1982) and Coombs' positive anaemia (Zulty and Kociba 1990).

Polyarthritis caused by mycoplasmas can be seen in many species including cattle, swine, dogs, goats, sheep and rats, and an erosive arthritis can develop (Walker 1999). In the veterinary literature, feline mycoplasma-associated polyarthritis is described as a non-erosive form (Abercromby 1994). This statement is based upon the publication of Moise et al (1982). The cat described did not show significant bone lesions on the radiograph, but subchondral erosions of the bone were found at post-mortem examination. This cat also had fever, mild anaemia, bilirubinuria, hypoproteinaemia, leukocytosis and kidney failure due to glomerulonephritis; clinical signs also seen in our cat. It did not have lymphadenopathy, but an enlargement of the regional lymph nodes was seen in experimentally infected cats.

The ability of mycoplasmas to trigger autoimmune responses is an area of active research in human medicine (Baseman and Tully 1997). Cold agglutinins can be found in 50% of human patients with *M pneumoniae* infection. The mechanism by which the autoimmune disease occurs is still controversial (Hahn 2005). Signs usually appear 2–3 weeks after infection starts. Extra- and intravascular haemolysis (Gehrs and

Friedberg 2002) and thrombocytopenia (Venkatesan et al 1996) are possible. Renal complications (interstitial nephritis and glomerulonephritis) are seen in less than 10% of human patients with atypical pneumonia caused by *M pneumoniae* (Sauter et al 1989). They are thought to be of post infectious aetiology associated with intravascular accumulation of immunoglobulins (Van Westrhenen et al 1998). Autoantibodies causing haemolytic anaemia can be seen in cats with *M haemofelis* infection (Zulty and Kociba 1990). We hypothesise that the Coombs' positive anaemia, thrombocytopenia and the renal failure of our cat could have been a direct sequel of mycoplasma infection. It is also possible that our cat was producing antibodies against red cell precursors, as only slight regeneration was seen for a short period.

Many signs in our cat are typical features seen in immune-mediated diseases such as systemic lupus erythematosus (SLE) or rheumatoid arthritis (Pederson and Barlough 1991, May and Bennett 1994). The non-regenerative anaemia diagnosed at the first day of presentation could have been caused by a pre-existing immune-mediated disease. The antinuclear antibody titre, a hallmark of SLE was negative, but seronegative cases of SLE have been described in other species (Pederson and Barlough 1991). It is possible, that the mycoplasma infection was secondary. The secondary infection would then explain the erosive polyarthritis not usually seen in SLE. As the histopathological examination of the synovial membrane showed no inflammatory infiltrates, rheumatoid arthritis as a pre-existing disease seems unlikely. Periosteal new bone formation, typical in the proliferative form of feline chronic progressive polyarthritis was not seen.

The atmospheric requirement for the recovery of mycoplasmas from synovial fluid was unusual in this case, as primary isolation of *M gateae* was only successful under strict anaerobic conditions. Although feline mycoplasmas are facultative anaerobes they usually favour a low-redox, enhanced CO₂ atmosphere at isolation (Whitford and Lingsweiler 1994). The routine examination for feline mycoplasmas would have given a false negative result in our case.

As a conclusion *M gateae* as a primary or secondary pathogen might play an important role in feline polyarthritis (erosive or non-erosive) and additional cultivation of the synovial fluid under strict anaerobic conditions is strongly recommended in cases where the routine bacteriological examination including mycoplasmas is

negative. Enrofloxacin, effective against many mycoplasmas in vitro (Bauditz 1987), was able to eliminate the pathogen from the joints and a rapid improvement of clinical signs was seen within 2 days of treatment. Whether *M. gateae* triggered an immune-mediated disease with Coombs' positive haemolytic anaemia, thrombocytopenia and glomerulonephritis or the signs were the consequence of a pre-existing immune-mediated disease could not be determined.

References

- Abercromby R (1994) Infective arthritis. In: Houlton J, Collinson R (eds), *BSAVA. Manual of Small Animal Arthrology* (1st edn). Cheltenham: British Small Animal Veterinary Association, pp. 100–112.
- Baseman JB, Tully JG (1997) Mycoplasmas: sophisticated, re-emerging, and burdened by their notoriety. *Emerging Infectious Diseases* **3**, 21–32.
- Bauditz R (1987) Results of clinical studies with Baytril in dogs and cats. *Veterinary Medical Review* **2**, 137–140.
- Bennett D, Nash AS (1988) Feline immune-based polyarthritis: a study of thirty-one cases. *Journal of Small Animal Practice* **29**, 501–523.
- Blackmore DK, Hill A, Jackson OF (1971) The incidence of mycoplasma in pet and colony maintained cats. *Journal of Small Animal Practice* **12**, 207–216.
- Carr AP, Michels G (1997) Identifying non-infectious erosive arthritis in dogs and cats. *Veterinary Medicine* **92**, 804–810.
- Carro T (1994) Polyarthritis in cats. *The Compendium (Small Animal)* **16**, 57–67.
- Carter GR, Wise DJ (2004) Mycoplasmas. In: Carter GR, Wise DJ (eds), *Essentials of Veterinary Bacteriology and Mycology* (6th edn). Ames: Iowa State Press, pp. 171–175.
- Foster SF, Martin P, Allan GS, Barrs VR, Malik R (2004) Lower respiratory tract infections in cats: 21 cases (1995–2000). *Journal of Feline Medicine and Surgery* **6**, 167–180.
- Gehrs BC, Friedberg RC (2002) Autoimmune hemolytic anemia. *American Journal of Hematology* **69**, 258–271.
- Gray LD, Ketring KL, Tang YW (2005) Clinical use of 16S rRNA sequencing to identify *Mycoplasma felis* and *M. gateae* associated with feline ulcerative keratitis. *Journal of Clinical Microbiology* **43**, 3431–3434.
- Hahn H (2005) Mykoplasmen und ureaplasmen. In: Hahn H, Falke D, Kaufmann SHE, Ullmann U (eds), *Medizinische Mikrobiologie und Infektiologie* (5th edn). Berlin-Heidelberg: Springer, pp. 422–426.
- Haesebrouck F, Devriese LA, Van Rijssen B, Cox E (1990) Incidence and significance of isolation of *Mycoplasma felis* from conjunctival swabs of cats. *Veterinary Microbiology* **26**, 95–101.
- Hooper PT, Ireland LA, Carter A (1985) Mycoplasma polyarthritis in a cat with probable severe immune deficiency. *Australian Veterinary Journal* **62**, 352.
- Jacques D, Cauzinille L, Bouvy B, Dupre G (2002) A retrospective study of 40 dogs with polyarthritis. *Veterinary Surgery* **31**, 428–434.
- Malik R, Love DN, Hunt GB, Canfield PJ, Taylor V (1991) Pyothorax associated with *Mycoplasma species* in a kitten. *Journal of Small Animal Practice* **32**, 31–34.
- May C, Bennett D (1994) Immune mediated arthritides. In: Houlton J, Collinson R (eds), *BSAVA. Manual of Small Animal Arthrology* (1st edn). Cheltenham: British Small Animal Veterinary Association, pp. 86–99.
- Moise NS, Crissman JW, Fairbrother JF, Baldwin C (1982) *Mycoplasma gateae* arthritis and tenosynovitis in cats: case report and experimental reproduction of the disease. *American Journal of Veterinary Research* **44**, 16–21.
- Pederson NC (1983) Joint diseases of dogs and cats. In: Ettinger SJ (ed), *Textbook of Veterinary Internal Medicine* (2nd edn). Philadelphia: W.B. Saunders, pp. 2187–2236.
- Pederson NC, Barlough JE (1991) Systemic lupus erythematosus in the cat. *Feline Practice* **19**, 5–13.
- Randolph JF, Moise NS, Scarlett JM, Shin SJ, Blue JT, Corbett JR (1993) Prevalence of mycoplasma and ureaplasma recovery from tracheobronchial lavages and of mycoplasma recovery from pharyngeal swab specimens in cats with or without pulmonary disease. *American Journal of Veterinary Research* **54**, 897–900.
- Rosengarten R, Yogev D (1996) Variant colony surface antigenic phenotypes within mycoplasma strain populations: implications for species identification and strain standardization. *Journal of Clinical Microbiology* **34**, 149–158.
- Sauter G, Heim M, Engelhardt D (1989) Atypische Pneumonie verbunden mit einer akuten Hämolyse bei einem 33jährigen Patienten. *Internist* **30**, 120–123.
- Spergser J, Aurich C, Aurich JE, Rosengarten R (2002) High prevalence of mycoplasmas in the genital tract of asymptomatic stallions in Austria. *Veterinary Microbiology* **87**, 119–129.
- Tan RJS, Lim EW, Ishak B (1977) Ecology of mycoplasmas in clinically healthy cats. *Australian Veterinary Journal* **53**, 515–518.
- Westrhenen Van, Weening JJ, Krediet RT (1998) Pneumonia and glomerulonephritis caused by *Mycoplasma pneumoniae*. *Nephrology Dialysis Transplantation* **13**, 3208–3211.
- Venkatesan P, Patel V, Collingham KE, Ellis CJ (1996) Fatal thrombocytopenia associated with *Mycoplasma pneumoniae* infection. *Journal of Infection* **33**, 115–117.
- Walker RL (1999) Mollicutes. In: Hirsh DC, Zee YC (eds), *Veterinary Microbiology* (1st edn). Malden: Blackwell Science, pp. 165–172.
- Whitford HW, Lingsweiler SW (1994) Canine, feline and equine mycoplasmas. In: Whitford HW, Rosenbusch RF, Lauerman LH (eds), *Mycoplasmosis in Animals: Laboratory Diagnosis*. Ames: Iowa State University Press, pp. 134–140.
- Zulty JC, Kociba GJ (1990) Cold agglutinins in cats with haemobartonellosis. *Journal of the American Veterinary Medical Association* **196**, 907–910.