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Pancreatitis associated with *Mycoplasma felis* infection in a cat

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Abstract

Case summary A 7-year-old domestic shorthair cat was presented for periuria, apathy, fever, inappetence, diarrhoea and vomiting. A complete blood count and biochemistry analysis revealed severe thrombocytopenia, severe azotaemia, moderate panhypoproteinemia, mildly elevated DGGR lipase activity and mildly elevated liver enzyme activity. Abdominal ultrasound showed a hypoechoic pancreas with surrounding hyperechoic fat demonstrating dirty shadowing and ascites (protein-poor transudate). The cat was treated medically for pancreatitis with fluid therapy, antiemetics and pain medication. During the hospitalisation period, the cat developed severe anaemia and received multiple whole blood transfusions yet showed no signs of clinical improvement. A repeat ultrasound examination performed after 8 days showed progressive pancreatic lesions and ongoing ascites. Analysis of the free abdominal fluid revealed neutrophilic inflammation despite low protein and cell concentration, with the presence of numerous very small, coccoid, basophilic inclusions within neutrophils, raising the concern for a septic peritonitis due to *Mycoplasma* species. Quantitative PCR (qPCR) confirmed the presence of *Mycoplasma felis*. After 10 days of hospitalisation, the cat developed refractory septic shock and was euthanased. Necropsy revealed severe necrotising pancreatitis with systemic changes consistent with sepsis and microthrombi. qPCR testing for *M felis* in pancreatic tissue also yielded a positive result.

Relevance and novel information Although pancreatitis is a common disease in cats, this case report presents the first documented occurrence of *M felis* as the suspected primary pathogen causing pancreatitis in a cat.

Keywords: Disseminated intravascular coagulation; necrotising pancreatitis; PCR; sepsis

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Introduction

Mycoplasma felis belongs to the class Mollicutes and is a prokaryotic organism without a cell wall.[1,2] It is a commensal bacterium of the respiratory and oral mucus membranes of cats but can invade deeper tissues in cases of host barrier disruption or secondary to immunosuppression.[1] *M felis* is transmitted through direct contact (respiratory, sexual) or fomite transmission in overcrowded environments.[1]

It commonly leads to conjunctivitis and upper respiratory tract signs, such as nasal discharge and sneezing.[3,4] Occasionally, it has been identified as the primary pathogen in conditions such as polyarthritis, meningoencephalitis, pneumonia and epididymitis-orchitis.[5–15]

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Table 1 Complete blood count profiles

	Day 1	Day 3	Day 8	Day 9	Reference interval	Unit
Parameter						
Haematocrit	0.31	**0.16**	**0.15**	**0.17**	0.28–0.47	l/l
MCV	44	46	43	44	38–52	fl
MCHC	**354**	337	347	350	310–350	pg
Reticulocytes	**3**	**4**	9	9	5–61	$\times 10^9$/l
Thrombocytes (confirmed by manual estimation)	**17**	**32**	**41**	**48**	180–520	$\times 10^9$/l
Leucocytes	7.59	12.86	**4.04**	**18.73**	6.5–15.4	$\times 10^9$/l
Manual differentiation						
Normoblasts	0	0	**0.02**	0	0	$\times 10^9$/l
Metamyelocytes	0	0	0	**0.28**	0	$\times 10^9$/l
Band neutrophils	0.27	**0.71**	**2.26**	**6.46**	0–0.3	$\times 10^9$/l
Segmented neutrophils	6.45	11.64	**1.09**	10.21	2.5–12.5	$\times 10^9$/l
Lymphocytes	**0.53**	0.26	**0.48**	1.4	1.5–7.0	$\times 10^9$/l
Monocytes	0.15	0.19	0.18	0.28	0–0.85	$\times 10^9$/l
Eosinophils	0.19	0.00	0.02	0	0–1.5	$\times 10^9$/l
Basophils	0.00	0.06	0.00	0	0–0.04	$\times 10^9$/l
Neutrophil toxicity		+	+++	+++		

Values in bold are outside the reference interval.
MCHC = mean cell haemoglobin concentration; MCV = mean cell volume, + = mild, +++ = severe

A diagnosis of *M felis* can be challenging, as they are smaller than usual bacteria and thus difficult to reliably identify cytologically. PCR is the diagnostic method of choice.[1,2,16]

The following case report describes *M felis* as the suspected primary pathogen in a cat with severe, acute, necrotising pancreatitis.

Case description

A 7-year-old male neutered domestic shorthair cat was referred to the Small Animal Hospital at the Vetsuisse Faculty, University of Bern, Switzerland due to a 7-day history of periuria, apathy, fever, inappetence, diarrhoea and vomiting. Before referral, the cat had been examined twice by the primary veterinarian and received symptomatic treatment (subcutaneous fluids, amoxicillin and metoclopramide). During the second visit, a chemistry profile showed azotaemia, an increase in liver enzyme activity and hyperbilirubinaemia.

Upon initial presentation at the Small Animal Hospital, the cat was found to be in a reduced general condition. Multiple haematomas were identified on both of the cat's front limbs as well as the right lateral thoracic wall. The cat also showed clinical signs indicative of cranial abdominal pain.

A complete blood count (CBC) showed severe thrombocytopenia and moderate lymphopenia (Table 1). A plasma biochemical analysis revealed electrolyte imbalances, moderate panhypoproteinemia, severe azotaemia, mild hyperbilirubinemia, mildly increased liver enzyme activities and mild increases in 1,2-*O*-dilauryl-*rac*-glycero-3-glutaric acid-(6′-mehylresorufin) ester (DGGR) lipase activity and serum amyloid A (Table 2). Coagulation tests, prothrombin time and activated partial thromboplastin time were mildly prolonged. Urinalysis of a free catch urine sample showed isosthenuria and an unremarkable dipstick (Combur Test; Cobas).

Abdominal ultrasound revealed hypoechogenicity of the pancreatic parenchyma, accompanied by surrounding hyperechoic fat and dirty shadowing (Figure 1). The stomach wall appeared thickened, specifically affecting the submucosal layer with otherwise preserved wall layering. The jejunal lymph nodes were mildly enlarged, and a mild amount of free peritoneal fluid was noted. There was mild bilateral renal pyelectasia, reduced corticomedullary differentiation and mild right renomegaly. Fine-needle aspirations of abnormal organs on ultrasound were not performed due to the thrombocytopenia.

During the work-up of fever, thoracic radiographs were performed and revealed a generalised cardiomegaly, a multifocal patchy alveolar lung pattern affecting the cranial lung lobes and mild pleural effusion.

A cytological analysis of the peritoneal effusion revealed a protein-poor transudate with low cellularity (total protein 22 g/l, total nucleated cell count 0.36×10^9/l), composed of a mixed population of macrophages/mesothelial cells and non-degenerate neutrophils. The primary differential for the pure transudate was fluid overload in combination with hypoalbuminemia. Other differentials, such as pre-sinusoidal hypertension (eg, portal vein thrombosis) or non-exfoliating neoplasia, were considered although none of these differentials could be definitively confirmed.

Table 2 Plasma biochemistry profiles

Parameter	Day 1	Day 3	Day 7	Reference interval	Unit
Na	**139**	154	151	147–157	mmol/l
K	**3.58**	4.85	3.63	3.7–5.3	mmol/l
Cl	**108**	**127**	115	112–124	mmol/l
Ca	1.89			2.22–2.92	mmol/l
P	**2.27**			0.82–1.91	mmol/l
Glucose	**5.97**	**10.55**	**9.5**	3.17–5.71	mmol/l
Cholesterol	2.48			2.09–4.52	mmol/l
Triglycerides	0.50			0.34–1.30	mmol/l
Total proteins	**47.4**			63–80	g/l
Albumin	**22.2**	**15**	**14.1**	30–42	g/l
Globulins	**25.2**			26–42	g/l
BUN	**49.3**			6.5–12.2	mmol/l
Creatinine	**616**	**199**	110	52–138	umol/l
Bilirubin	**26.8**	**53.6**	**19.8**	0–6.2	umol/l
ALT	**171**			12–77	U/l
AP	12			0–93	U/l
ASAT	**162**			12–61	U/l
CK	**6167**			0–596	U/l
gGT	<3			<3	U/l
GLDH	**17**			0–3	U/l
Lipase (DGGR)	**83**			8– 26	U/l
Serum amyloid A	**19**	**92.7**	**129**	<8	µg/ml

Values in bold are outside the reference interval.
ALT = alanine transaminase; AP = alkaline phosphatase; ASAT = aspartate aminotransferase; BUN = blood urea nitrogen; Ca = calcium; CK = creatinine kinase; Cl = chloride; DGGR = 1,2-*O*-dilauryl-*rac*-glycero-3-glutaric acid-(6′-methylresorufin) ester; gGT = gamma-glutamyl transferase; GLDH = glutamate dehydrogenase; K = potassium; Na = sodium; P = phosphate

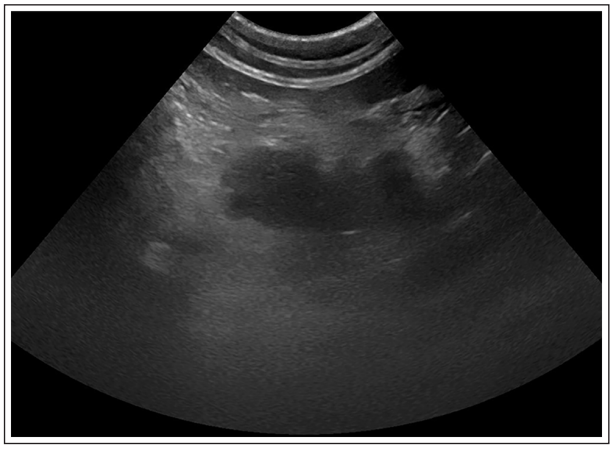

Figure 1 Abdominal ultrasound image showing a hypoechoic pancreas surrounded by hyperechoic fat demonstrating dirty shadowing at day 1 of hospitalisation

Several tests for infectious diseases, including feline immunodeficiency virus/feline leukaemia virus SNAP test (SNAP FIV/FeLV Combo Test; IDEXX), toxoplasma serology and feline coronavirus reverse-transcription PCR of peritoneal effusion yielded negative results.

Based on the available data, a suspected diagnosis of severe pancreatitis with subsequent systemic inflammatory response syndrome and disseminated intravascular coagulation (DIC) was made. The initial azotaemia was fluid-responsive and quickly improved within a day.

During the first 3 days of hospitalisation, the cat developed an acute, non-regenerative, normocytic, normochromic anaemia, as well as worsening of the hypoalbuminaemia (Tables 1 and 2). The cat was treated with three whole blood transfusions (from related cats, living in the same household), one plasma transfusion, one transfusion of human albumin (Albumin CSL 20%; CSL Behring) and intravenous amino acid infusion (Aminoven 15%; Fresenius Kabi).

Additional treatment for pancreatitis and coagulopathy consisted of intravenous crystalloid fluids (Ringeracetat; Fresenius Kabi), buprenorphine (Bupaq; Streuli Tiergesundheit) 0.02 mg/kg IV q8h, ondansetron (Ondansetron; Labatec) 0.3 mg/kg IV q8h, maropitant (Cerenia; Zoetis) 1 mg/kg IV q24h, mirtazapine ear cream 0.75 mg q24h (compounded product), vitamin K (Konakion; Roche) 2 mg/kg IV q12h and tranexamic acid (Cyklokapron; Pfizer) 10 mg/kg IV q8h. Because of the suspicion of immune-mediated thrombocytopenia, the cat was treated with dexamethasone (Dexafast; Graeub) 0.2 mg/kg IV q24h.

On day 3 of hospitalisation, the CBC showed an increase of neutrophil concentration towards the upper end of the reference interval with mild toxicity and left shift. Antimicrobial therapy with ampicillin-sulbactam

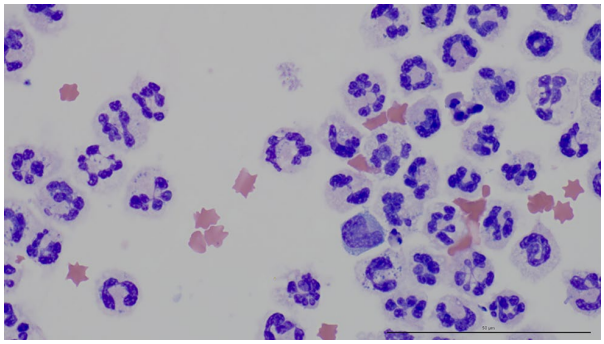

Figure 2 Photomicrograph of a cytocentrifuge preparation of peritoneal effusion from a cat, showing neutrophilic inflammation with multiple small, coccoid, intracellular organisms, confirmed as *Mycoplasma felis* (total protein 26 g/l, cell count 2.0×10^9/l). Wright–Giemsa stain, × 100 oil objective. Bar = 50 µm

(Ampicillin/Sulbactam; Fresenius Kabi) 30 mg/kg IV q12h was initiated.

Despite medical treatment, the cat's clinical signs appeared to worsen, which prompted a repeat abdominal ultrasound examination on day 8 of hospitalisation. This revealed increased dirty shadowing of the peripancreatic fat and an ill-defined hypoechoic pancreas. The changes observed in the stomach and jejunal lymph nodes remained stable, but additional diffuse thickening of the hypoechoic jejunal loops was observed. The bilateral renal pyelectasia slightly decreased. In addition, there was an increased amount of free peritoneal fluid. Concurrently, a CBC indicated a degenerative left shift and severe toxicity. Therefore, treatment with marbofloxacin (Marbocyl; Vétoquinol) 2 mg/kg IV q24h was initiated.

The peritoneal free fluid was re-aspirated, revealing low cellularity (2×10^9/l), low total protein (26 g/l) and a high percentage of neutrophils (89%). Most neutrophils contained small, coccoid, basophilic inclusions, raising the suspicion of *Mycoplasma* species infection (Figure 2). The DGGR lipase activity in the peritoneal effusion was 1167 U/l, which was >30 times higher than the concurrent plasma concentration. Quantitative PCR (qPCR) testing confirmed the presence of *M felis* in the peritoneal effusion (cycle threshold (CT) value: 21.8). Subsequently submitted blood samples from the cat tested positive for *Mycoplasma hemofelis* and '*Candidatus* Mycoplasma haemominutum', but negative for '*Candidatus* Mycoplasma turiciensis'. Routine bacterial culture was not performed, as the cat was already under double antibiotic therapy. Doxycycline (DoxyCat; Biokema SA) 10 mg/kg PO q24h was added to the treatment with the specific aim of targeting *M felis*.

On day 10 of hospitalisation, the cat's general condition further deteriorated, and there was no response to fluid and vasoconstrictor treatment for hypotension. As a result, the cat was euthanased, and the owners gave consent for a post-mortem examination.

Necropsy confirmed a severe, multifocal, chronic-active necrotising pancreatitis with adipose tissue necrosis (Figure 3). Additional lesions included peritonitis, enteritis, myocarditis and pulmonary haemorrhages (see figure in the supplementary material). The changes in the peritoneum, small intestine, lungs and heart were consistent with secondary complications resulting from pancreatitis, DIC and sepsis.

Post-mortem qPCR testing of the pancreas confirmed the presence of *M felis* (CT value 28), but the myocardium tested negative.

Discussion

This report describes a case of acute necrotising pancreatitis accompanied by DIC and sepsis, attributed to *M felis* infection. Cytological examination of the ascitic fluid raised the suspicion of *Mycoplasma* species within neutrophils, and qPCR testing confirmed the infection. The lipase activity in the ascitic fluid was significantly elevated (>30-fold increase compared with plasma), suggesting fluid formation secondary to severe pancreatitis.[17] A subsequent qPCR analysis of a pancreatic tissue sample also yielded a positive result for *M felis*, supporting the theory of pancreatic involvement. However, contamination from the ascitic fluid cannot be completely ruled out as a potential source of false-positive qPCR results in the pancreas.

Based on an extensive literature search conducted until 16 November 2023, using databases such as PubMed and Science Direct with the keywords 'Mycoplasma' and 'feline' or 'cat' and 'pancreatitis', this is the first published report documenting *M felis* as the suspected primary pathogen associated with acute necrotising pancreatitis in cats.

In human medicine, several case reports have described patients with acute pancreatitis associated with *Mycoplasma pneumoniae* infection.[18–25] The patients typically exhibited respiratory signs as the predominant clinical manifestation,[18,20] which were absent in the cat described here.

Notably, the cat had not been tested for Mycoplasma before visualisation of *Mycoplasma* species in the ascitic fluid. Therefore, it remains unclear whether the cat had a pre-existing subclinical *M felis* infection in the upper respiratory tract or urinary tract,[26] which subsequently disseminated to the pancreas during the disease course, or if *M felis* infection occurred at the onset of clinical signs.

The severe thrombocytopenia and prolonged clotting times are believed to be secondary to DIC resulting from necrotising pancreatitis. Immune-mediated thrombocytopenia secondary to systemic inflammation should also be considered. Although the hemotropic *Mycoplasma* species documented in this case are well known to be a primary cause of anaemia, positivity for hemotropic *Mycoplasma* species has also been shown to be associated

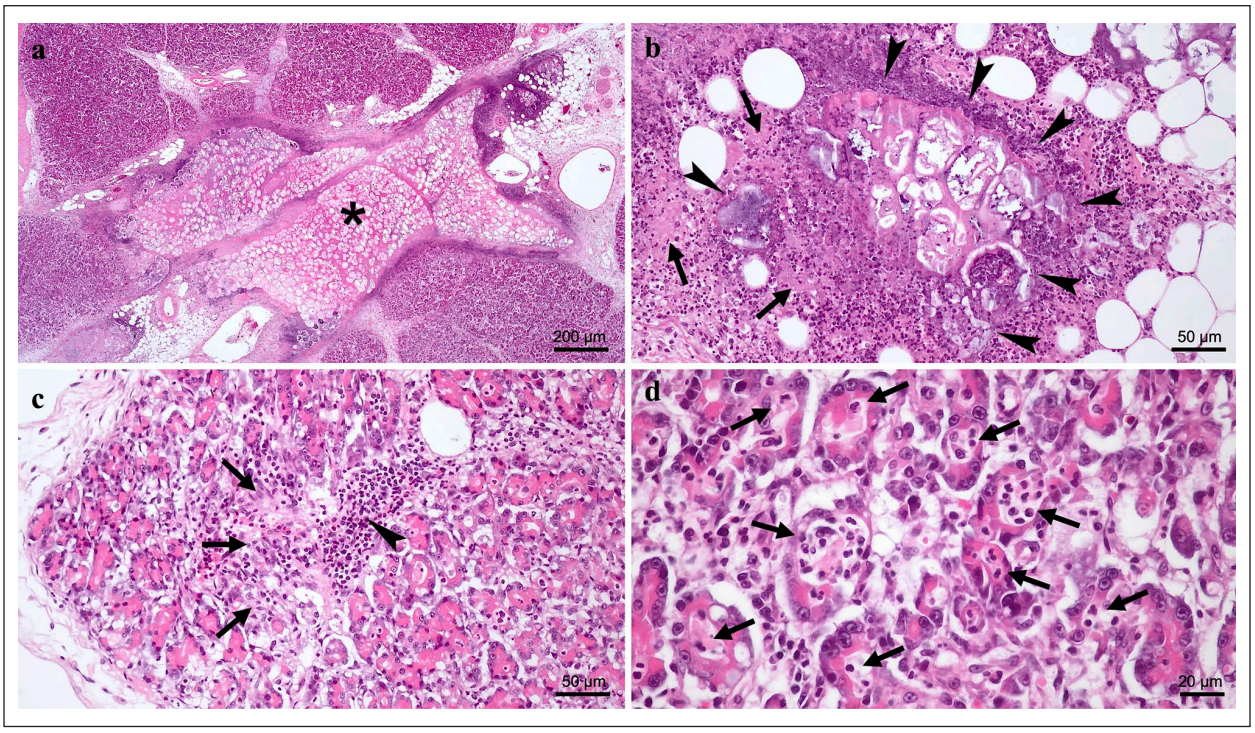

Figure 3 Histopathological features of necrotising pancreatitis (haematoxylin and eosin stain). (a) Focally extensive area of fat necrosis and saponification (asterisk). Magnification × 4. (b) Necrotic adipocytes surrounded by numerous, partially degenerated neutrophils and fewer macrophages (arrowheads), and areas of necrosis with cell debris (arrows). Magnification × 20. (c) Multifocal to coalescing areas of interstitial fibrosis (arrows) accompanied by interstitial infiltration of numerous neutrophils, lymphocytes and macrophages (arrowhead). Magnification × 20. (d) Multifocal infiltration of neutrophils into the acini with degeneration and necrosis of the pancreatic acinar cells (arrows). Magnification × 40

with lower platelet concentrations.[27] They might have therefore contributed to the severe thrombocytopenia.

During hospitalisation, the cat developed a non-regenerative normocytic, normochromic anaemia, suspected to be of multifactorial cause. Hyperbilirubinaemia could prompt the suspicion of immune-mediated haemolytic anaemia (IMHA), but bilirubin concentrations were already elevated on day 1 of hospitalisation with normal haematocrit. Therefore, criteria for the diagnosis of IMHA based on the American College of Veterinary Internal Medicine (ACVIM) consensus guidelines were not met.[28] Other potential causes for the anaemia include thrombocytopenia leading to unnoticed haemorrhage, as well as inflammatory disease, which can cause oxidative injury to erythrocytes and dampens the development of a regenerative response. After three whole blood transfusions, the cat tested positive for *M hemofelis* and *Candidatus* M haemominutum, which, in theory, could have caused the anaemia itself. The cat was not initially screened for haemotropic *Mycoplasma* species and therefore it remains unclear whether it was initially infected with these species, or whether they were transmitted through the whole blood transfusions.

For this specific case, it is imperative to differentiate between haemotropic and non-haemotropic *Mycoplasma* species. Haemotropic Mycoplasmas affect red blood cells and can be transmitted by blood transfusions; however, *M felis* cannot.[2,29]

While a pre-existing infection with *M hemofelis* and *Candidatus* M haemominotum could explain the anaemia and thrombocytopenia, it does not account for the pancreatitis. The pancreatitis, however, can be explained by the *M felis* infection, as detected by qPCR in the second peritoneal fluid analysis and the pancreas.

The cause for the initial severe azotaemia remains unclear but is most likely multifactorial. Considering the isosthenuria, pre-renal azotaemia seems unlikely. Acute pancreatitis is known to potentially cause acute kidney injury (AKI),[30,31] although it seems atypical that AKI occurs before the pancreatitis has reached its clinical peak. Another hypothesis to consider would be that the initial azotaemia was due to obstructive ureterolithiasis, which spontaneously resolved under fluid therapy. However, on initial abdominal ultrasound, only a mild pyelectasia with no clear signs of obstructive ureteroliths were noted. A mild pyelectasia can also occur with bacterial pyelonephritis or secondary to fluid therapy.

In response to a development of a left shift and toxicity in the CBC on day 3 of hospitalisation, antimicrobial therapy with ampicillin-sulbactam, a broad-spectrum

antibiotic, was initiated. Beta-lactamase inhibitors inhibit bacterial cell wall synthesis and are therefore ineffective against bacteria lacking a cell wall.[32] Antimicrobials that are effective against *Mycoplasma* species were started only on day 8 of hospitalisation. This delay in effective antimicrobial treatment most likely contributed to the exacerbation of clinical signs. Routine bacterial culture of the ascites was not performed, as the cat was already under antimicrobial treatment against the identified pathogen.

Conclusions

This report presents the first documented case of necrotising pancreatitis associated with *M felis* infection in a cat. The pancreatitis in this case was severe and progressively worsened, presumably leading to sepsis. Despite intensive therapy, the cat's clinical condition deteriorated further, resulting in the development of refractory septic shock. Consequently, *M felis* infection should be considered as a potential, albeit rare, underlying cause of severe pancreatitis in cats.

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Supplementary material The following file is available as supplementary material: Figure: Additional histopathological findings (haematoxylin and eosin).

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers) for all procedure(s) undertaken (prospective or retrospective studies). No animals or humans are identifiable within this publication, and therefore additional informed consent for publication was not required.

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