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Necrotising fasciitis, a potential threat following conservative treatment of a leucopenic cat: a case report

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ABSTRACT: An eight-month-old, not vaccinated, intact male domestic shorthair cat from a multi-cat household was presented at the clinic because of fever, inappetence and listlessness. Although leucopenic, it was first treated with antibiotics and subcutaneous fluid administration. After several days of hospitalisation with only symptomatic treatment, it developed a vast area of skin necrosis and was consequently euthanised. Necropsy was performed revealing morphological lesions consistent with necrotising fasciitis (NF). Three multidrug resistant bacteria were isolated from the tissue.

Keywords: cat; necrotising fasciitis; immunosuppression; ESBL; MRSH; HLAR

List of abbreviations
ESBL = extended spectrum beta lactamase-producing *E. coli*, **HE** = haematoxylin and eosin, **HLAR** = high-level aminoglycoside-resistant *Enterococcus* sp., **MRSH** = methicillin-resistant *Staphylococcus haemolyticus*, **NF** = necrotising fasciitis

Necrotising fasciitis (NF) is a rapidly spreading and potentially life-threatening bacterial infection of the subcutaneous and fascial tissues, minor skin injuries being the usual portal of entry. Signs of infection appear a few days following infection and include localised erythema, oedema and pain of the affected area (Naidoo et al. 2005). The diagnosis of NF is based on the clinical signs, surgical findings, histopathology results and positive culture of a recognised aetiological agent (Liu et al. 2005). The most important component of treatment is aggressive surgical excision of all necrotic tissue to remove the bacterial nidus and to prevent further spread along fascial planes (Berube et al. 2010). Delay in surgical debridement increases the mortality rate significantly (Burch et al. 2007). Medical therapy in the absence of surgical debridement is unsuccessful due to poor antibiotic delivery to the affected area and the continued production of bacterial toxins (Naidoo et al. 2005; Hess 2009).

Although NF has been widely reported in human medicine, with an incidence of one to five cases per 100 000 people (Worth et al. 2005), there are no records of its incidence in veterinary medicine. It is a rare condition, although, in the last two decades, accounts of its occurrence are emerging, mostly in dogs (Miller et al. 1996; Prescott et al. 1997; DeWinter et al. 1999; Jenkins et al. 2001; Naidoo et al. 2005; Worth et al. 2005; Kulendra and Corr 2008; Plavec et al. 2008; Weese et al. 2009; Csiszer et al. 2010), but also in cats (Brachelente et al. 2007; Pesavento et al. 2007; Sura et al. 2008; Hess 2009; Berube et al. 2010) and other animals (Zappulli et al. 2005; Bishop et al. 2007; Allender et al. 2009; Bancroft-Hunt et al. 2010). The majority of cases in veterinary medicine are caused by *Streptococcus canis* (Miller et al. 1996; Prescott et al. 1997; DeWinter et al. 1999; Jenkins et al. 2001; Naidoo et al. 2005; Kulendra and Corr 2008). Polymicrobial NF, which is very common in human medicine, has been reported only once – in a cat with a necrotising soft tissue infection of the genital and anorectal regions (Berube et al. 2010). Here we report NF of the tho-

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racic region caused by an extended spectrum beta lactamase-producing *E. coli* (ESBL), methicillin-resistant *Staphylococcus haemolyticus* (MRSH) and a high-level aminoglycoside-resistant *Enterococcus* sp. (HLAR) that occurred after conservative treatment of a leucopenic cat.

Case description

An eight-month old, not vaccinated, 3.64 kg intact male domestic shorthair cat from a multicat household was examined due to acute onset of fever, inappetence and listlessness. The cat was an indoor-outdoor cat; the other household cats showed no signs of illness.

At physical examination body temperature was 40.3 °C, femoral pulse 160 beats per minute and respiration rate 28 breaths per minute. The cat was approximately 5% dehydrated; no other abnormalities were observed. The cat was first treated using subcutaneous fluid therapy with lactated Ringer's solution 100 ml *s.c.*, amoxicillin with clavulanic acid at 20 mg/kg/12 h *s.c.* (Synulox®, Pfizer), gentamicin at 4 mg/kg q 24 h *i.m.* (Gentamicin®, Krka) and metoclopramide at 0.2 mg/kg q 8 h *s.c.* (Reglan® Alkaloid). After nine hours the temperature dropped to 39.5 °C but on the next day it rose again to 40.5 °C.

Abnormalities found at that time on complete blood count and serum examination were leucopenia 0.46 × 10^9/l (reference range 5.5–19.5 × 10^9/l), with neutropenia 0.04 × 10^9/l (reference range 2.5–12.5 × 10^9/l), lymphopenia 0.38 × 10^9/l (reference range 1.5–7 × 10^9/l) and monocytopenia 0.03 × 10^9/l (reference range 0.1–0.85 × 10^9/l), thrombocytopenia 139 × 10^9/l (reference range 300–700 × 10^9/l) and hypokalaemia 3.38 mmol/l (reference range 4.0–4.5 mmol/l). Faeces were collected via rectal swab; an in-house parvovirus test (FASTest® PARVO Strip; Megacor, Austria) was negative. Further diagnostic evaluation was not performed because of significant financial constraints. Amoxicillin with clavulanic acid at 20 mg/kg/12 h *i.v.* (Augmentin®, GlaxoSmithKline) was provided instead of Synulox®. Metronidazole at 10 mg/kg q 12 h *i.v.* (Efloran®, Krka) and meloxicam at 0.1 mg/kg q 24 h *i.v.* (Metacam®, Bohringer Ingelheim) were added, and intravenous fluid therapy, supplemented with potassium chloride (30 mEq/l) in lactated Ringer's solution at 5 ml/kg/h, was started.

From the second through fifth day of hospitalisation, the patient remained lethargic and febrile at 40.0–40.7 °C. During that time it was force-fed and remained normotensive with a systolic blood pressure of 120 mmHg. On the fifth day the numbers of leucocytes and neutrophils were still low (3.15 × 10^9/l and 1.6 ×10^9/l) but, on the sixth day, increased to normal (leucocytes 9.65 × 10^9/l; neutrophils 7.6 × 10^9/l) with a left shift, but the haematocrit fell to 0.25 l/l (reference range 0.30–0.45 l/l) from the initial 0.35 l/l. The cat then started eating on its own. Its temperature on the sixth day was 38.2 °C and rose to 39.0 °C in the evening of the seventh day. On the eighth day it reached 40.0 °C and soft-tissue swelling was noted in the left lateral thoracic region, where *s.c.* fluids were given. A fine needle aspiration yielded a small amount of suppurative material with large numbers of degenerate neutrophils and intracellular and extracellular, mostly coccoid, bacteria. Differential diagnosis dictated surgical exploration, but the cat was anaesthetised no sooner than eight hours afterwards, when owner permission was gained. A vast area of skin necrosis was noted and, at that stage, the owner decided to euthanise the cat and allowed necropsy.

On the fifth day of treatment, another cat from the same household was examined with similar clinical signs and with panleucopenia (leucocytes 0.29 × 10^9/l, neutrophils 0.07 × 10^9/l, lymphocytes 0.16 × 10^9/l and monocytes 0.02 × 10^9/l) and thrombocytopenia (PLT 134 × 10^9/l). The owner decided for euthanasia, but necropsy was not performed.

At necropsy of the original patient, an oval, well-demarcated area of skin necrosis, measuring 13 cm × 12 cm, was evident at the left costal region, extending along the costal margin (Figure 1). The necrotic skin was detached by a one to four centimetre wide zone of uncovered, inflamed and necrotic subcutis and muscles. The necrotic skin was easily removed from the underlying subcutis which was severely and diffusely thickened with yellow green, dense, foul-smelling exudate forming several pockets filled with pus. The subcutaneous lymph nodes were moderately enlarged. Besides these lesions, a subcutaneous haematoma in the right costal region, dilated heart chambers, severe myocardial anaemia, severe pulmonary oedema, mild pleural transudation, mild liver congestion, several linear mucosal haemorrhages of the middle jejunal segment, with moderate hyperplasia and congestion of the spleen, were presented. Blood in large blood vessels was thin and unclotted.

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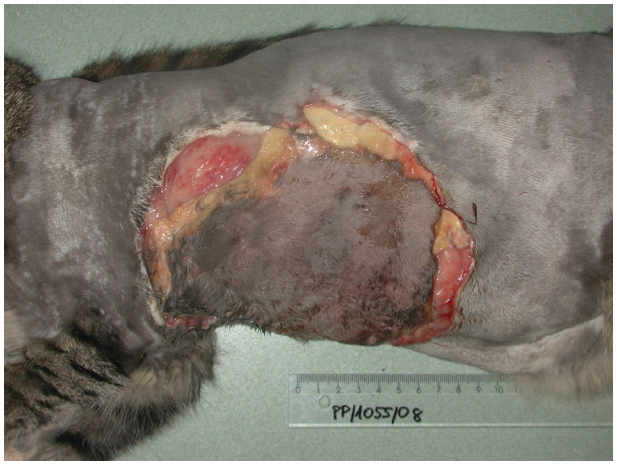

Figure 1. Deceased patient in right lateral recumbency. Extensive oval area of skin necrosis in the left costal region. Note the clear line of demarcation with purulent discharge

Representative specimens of the skin and organs were collected during necropsy and fixed in 10% neutral buffered formalin for 24 h, routinely embedded in paraffin, sectioned at 4 µm and stained with haematoxylin and eosin (HE).

Histopathological examination of the skin and subcutis revealed morphological lesions consistent with necrotising fasciitis. Epidermis, dermis and subcutis showed acute-to-subacute, massive coagulative necrosis, with parts of the epidermis missing. Deep dermis, subcutis and cutaneous muscles were infiltrated with degenerate neutrophils and macrophages, filling spaces between superficial and deep fascia, thus forming large, multifocal and coalescing accumulations of pus (Figure 2). Blood vessels in affected areas were dilated and thrombosed. Numerous small coccoid and fewer rod-shaped bacteria were evident in all skin layers and subcutis. Young granulation tissue and multifocal haemorrhages were found at the border of necrotic and inflamed tissue. Subcutaneous muscles and deep thoracic muscles in inflamed areas showed hyaline degeneration, coagulative necrosis and severe interstitial oedema. Regional lymph nodes of the affected skin areas exhibited marked reactive hyperplasia, while other lymph nodes showed prominent erythrophagocytosis.

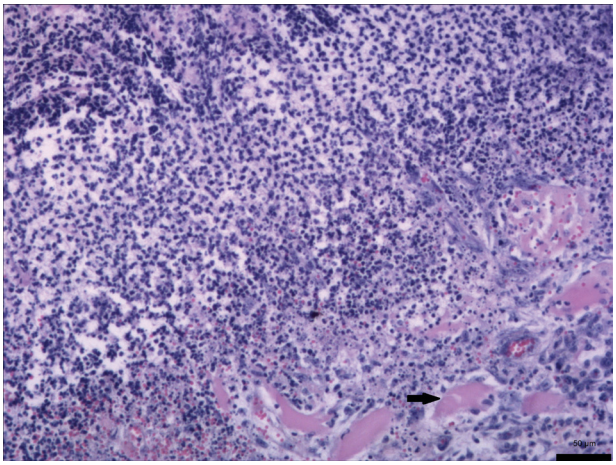

Figure 2. Histology of necrotic fasciitis: deep muscular fascia at the borderline with subcutaneous fat is necrotic and infiltrated with numerous neutrophils and macrophages, only fragments of muscle fibres are preserved (arrow). HE staining × 100; bar = 50 µm

Histopathological examination of other organs revealed acute tissue lesions related to the severe acute septic inflammation, such as cloudy swelling of the myocardium and the liver, hepatic sinusoidal monocytosis and lymphocytosis, severe acute pulmonary oedema with mild multifocal emphysema, marked reactive follicular and ellipsoid hyperplasia of the spleen, and small multifocal meningeal haemorrhages.

At necropsy, subcutaneous swabs and samples of the spleen, liver, small intestine, lungs and kidneys were taken for bacteriological examination. A sample of antibiotic suspension (amoxicillin with clavulanic acid – Synulox®) used during the therapy was taken in order to check potential bacterial contamination.

Bacteriology examinations were performed in Columbia agar supplemented with 5% of ovine blood and incubated at 37 °C under both aerobic and anaerobic conditions. *Staphylococcus haemolyticus*, *Enterococcus faecalis* and *E. coli* were isolated from subcutaneous swabs, but not from samples of internal organs. All isolated microorganisms were identified on the basis of colony morphology, Gram stain, and biochemical characteristics. Final confirmation was established with commercial biochemical sets (API System, BioMerieux, France) – API Staph for *Staphylococcus haemolyticus*, API Strep for *Enterococcus faecalis* and API20E for *E. coli*. Antibiotic susceptibility of isolated strains was determined by the microdilution method, using commercial antibiotic microplates, interpreted according to the CLSI guidelines. Sensititre GPALL1F panel (Trek, Diagnostic System) was used for testing *S. haemolyticus* and *E. faecalis* isolates and EUMVS2 for *E. coli*.

Screening tests for extended-spectrum β-lactamase (ESBL) activity were evaluated with respect to their susceptibility to cefpodoxime (10 µg), ceftazidime (30 µg), cefotaxime (30 µg) and ceftriaxone

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Table 1. Results of antibiotic susceptibility testing of bacterial isolates

Used factor	*Staphylococcus haemolyticus*		*Enterococcus* sp.	
Antimicrobials	MIC GPALL1F		MIC GPALL1F	
Chloramphenicol	4	S	8	S
Daptomycin	≤ 0.5	S	4	S
Gentamicin	8	I	> 16	R
Linezolid	≤ 1	S	2	S
Rifampin	≤ 0.5	S	> 4	R
Trimethoprim/ sulfamethoxazol	> 4/76	R	> 4/76	R
Quinupristin/ dalfopristin	≤ 0.5	S	4	R
Tetracycline	> 16	R	> 16	R
Erythromycin	> 4	R	> 8	R
Oxacillin indicator	> 4	MRS	> 4	
Cefoxitin indicator	> 6	MRS	> 6	
Ampicillin	> 8	R	> 8	R
Penicillin	> 8	R	> 8	R
Vancomycin	1	S	1	S
Levofloxacin	4	R	> 4	R
Tigecycline	0.5		0.5	
Moxifloxacin	1	I	> 4	
Clindamycin	≤ 0.5	S[a]	>2	
Streptomycin	> 1000		> 1000	HLAR
Ciprofloxacin	> 2	R	> 2	R
Nitrofurantoin	≤ 32	S	64	I
Gentamicin 500	≤ 500		> 500	HLAR

Results were evaluated according to CLSI, Performance Standards for Antimicrobial Susceptibility testing; (M100-S23, 2013)
MRS = methicillin-resistant *Staphylococcus*; HLAR = high-level aminoglycoside resistance [a]D test by microdilution negative (no inducible clindamycin resistance)

(30 µg) (BBL-Difco). Tests for ESBL were confirmed by E-test for cefotaxime/cefotaxime + clavulanic acid (CT/CTL) and ceftazidime/ceftazidime + clavulanic acid (TZ/TZL) (AB-Biodisk, Dalvagen, Solna, Sweden) and, additionally, by ESB1F panel (Trek, Diagnostic System). The reference microorganisms *E. coli* (ATCC 25922) and *Klebsiella pneumoniae* (NEQAS 7383/05, ESBL strain) were used as control strains. The results are shown in Tables 1 and 2. The *E. coli* isolate was further assigned to phylogenetic group B1 according to Clermont et al. (2000) and analysed for the presence of ESBL CTX-M group (Woodford 2010) and virulence factor genes, including fimbriae/adhesins (*fimH*, *crl*, *matA*, *papGI*, *papGII*, *papGIII*, *sfa*, *aaf*, *afa/dra*, *gaf*, *iha*, *bfp*, *bmaE*, *hra*), toxins/autotransporters (*hlyA*, *ehxA*, *cnfI*, *cnfII*, vtx_1, vtx_2, *cdt*, *hbp*, *sat*, *vat*, *picU*, *fluA*, *astA*), invasins (*ompA*, *ibeA*, *aslA*), iron acquisition systems (*iucD*, *iutA*, *iroN*, *fyuA/irp2*), protectins (*kpsMT*II, *iss*, *traT*), genotoxins (*usp*, *clbB*, *clbN*, *cdtB*, *cdtS*) and colicin/microcin V (*cvi*) (Johnson and Stell 2000; Johnson et al. 2003; Toth et al. 2003; Ewers et al. 2005; Girardeau et al. 2005; Johnson et al. 2008; Kemmett et al. 2013).

ESBL-producing *E. coli*, methicillin-resistant *Staphylococcus haemolyticus* (MRSH) and high-level aminoglycoside-resistant *Enterococcus sp.* (HLAR) were isolated from *subcutaneous* swabs. All three isolates were multidrug resistant bacteria. The sample of antibiotic suspension was sterile.

DISCUSSION AND CONCLUSIONS

NF is an uncommon but serious infection of the subcutaneous tissue and fascia with relative sparing of the skin and muscle, both of which may be infected secondarily (Cunningham et al. 2001). In humans, trauma, surgery and immunosuppression are predisposing factors (Brachelente et al. 2007), the latter being the possible explanation for the emergence of fasciitis in our patient. The definitive cause of leucopenia and neutropenia in the beginning of the clinical course of the disease remained undetermined in our case. The most frequently encountered causes of neutropenia in cats are non-bacterial infectious disease (fungal or viral), mostly due to feline parvovirus (FPV), feline leukaemia virus (FeLV) or feline immunodeficiency virus (FIV), followed by bacterial infection or overwhelming inflammation (Brown and Rogers 2001; Schnelle and Barger 2012). However, no gross or microscopic lesions of feline panleucopenia were seen in our case. Further, the in-house parvovirus test was also negative. Since virological examinations were not carried out, we cannot exclude other viral infections that could cause immunosuppression. Pesavento et al. (2007) described an outbreak of fatal *Streptococcus canis* NF in intensively housed shelter cats in which the role of an immunosuppressive condition or pathologically silent viral disease that preceded *Streptococcus* infection was also suspected.

The initial source of the infection has not been confirmed. There were no visible lesions, but the

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Table 2. Results of antibiotic susceptibility testing of bacterial isolates (*Escherichia coli)*

Antimicrobials	MIC EUMVS 2		Antimicrobials	MIC ESB1F	
Nalidixic acid	> 64	R	ampicillin	> 16	R
Florfenicol	4		piperacillin + tazobactam	≤ 4/4	
Chloramphenicol	4		cefotaxime	> 64	R
Gentamicin	32	R	cefotaxime + clavulanic acid	≤ 0.12/4	ESBL
Kanamycin	≤ 4	S	ceftazidime	4	ESBL
Streptomycin	64	R	ceftazidime + clavulanic acid	≤ 0.12/4	
Tetracycline	> 64	R	cefazolin	>16	R
Colistin	≤ 2	S	cefepime	4	
Sulphonamides	> 1024	R	cefoxitin	≤ 4	S
			cephalothin	> 16	R
			cefpodoxime	> 32	R
Trimethoprim	≤ 0.5	S	ceftriaxone	128	R
			ciprofloxacin	≤ 1	S
			imipenem	≤ 0.5	S
			meropenem	≤ 1	S

Results were evaluated according to CLSI, Performance Standards for Antimicrobial Susceptibility testing; Twenty-Third Informational Supplement (M100-S23, 2013)

antibiotic injection and subcutaneous fluid had been given in the affected area. Although skin contamination at injection sites (Fox et al. 1981) or even contaminated drugs may have been the source, the latter appears unlikely. The bottle of antibiotic was sterile and contamination of other drugs or fluids is less likely, since they are opened new for every patient. Subcutaneous fluids, which are convenient for slow replenishment of the mild and moderately dehydrated patient (Rosenfeld et al. 2012), are unacceptable in an intensive care setting (DiBartola and Bateman 2006). In practice, they are often administered to cats with panleucopenia because of financial limitations, as in our case. These cats may be severely dehydrated and, further, their compromised immune system may not fight off an infection that had been introduced subcutaneously. In one case of NF, subcutaneous fluids were administered to the cat without complications at the site of administration (Brachelente et al. 2007).

Nosocomial infections may be derived from exogenous microorganisms acquired by patients during their stay in hospital or from the endogenous flora of the patients. The main agents of nosocomial infections are Gram-positive cocci (staphylococci and enterococci), members of the Enterobacteriaceae, and non-fermentative Gram-negative bacilli (*Acinetobacter* spp. and *Pseudomonas* spp.) (Boerlin et al. 2007). When polymicrobial synergistic infection is identified in humans with NF, streptococci and enterobacteria are the most common co-isolates (Wong et al. 2003). In veterinary medicine only one case of polymicrobial necrotising soft tissue infection has been reported in a cat (Berube et al. 2010). This was a case of Fournier's gangrene, a specific type of necrotising fasciitis of the genital, perianal, and perineal regions of the body, in which the majority of cases are caused by normal flora of the lower gastrointestinal tract (Burch et al. 2007; Berube et al. 2010). The isolated bacteria, *Enterococcus faecium*, *Staphylococcus epidermidis* and *E. coli,* were similar to those in our case and these genera of bacteria were also isolated from duodenal juice aspirates of specific pathogen-free (SPF) cats (Sparkes et al. 1998). One of the isolates in our case, *Staphylococcus haemolyticus*, was not identified in the study of Sparkes et al. (1998), but had previously been isolated from cats with urinary tract infection, from an abscess (Igimi et al. 1994) and from 2.7% of 113 specimens of healthy cats, mostly from the hair coat (Cox et al. 1985). The *E. coli* isolate was assigned to phylogenetic group B1 and carried genes encoding the adhesins/fimbriae (*fimH*, *matA*, *crl*), the yersiniabactin iron-acquisition system fyuA/irp-2, which provides iron to the bacteria during infection and reduces the innate immune response of the host (Paauw et al. 2009), and the conjugal transfer surface

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exclusion protein (*traT*) involved in serum resistance. None of the screened toxin genes was detected. The low number of virulence genes is in accord with previous findings, suggesting that group B1 isolates are commensals and only infrequently cause extra-intestinal infections, except in immunocompromised hosts (Picard et al. 1999; Johnson et al. 2001; Johnson 2002; Branger et al. 2005). Thus, we consider that all the isolated bacteria originated from the resident microbiota of the patient, most probably from the gastrointestinal tract, although *S. haemolyticus* may have originated from the skin.

Moyaert et al. (2006) investigated the prevalence of acquired antimicrobial resistance in the resident intestinal microbiota of cats, and found 27.3% (the largest percentage of resistant isolates were isolated from hospitalised cats), 75.9%, 90.5% and 41.9% resistant isolates of *E. coli*, *E. faecalis, E. faecium* and *Streptococcus canis*, respectively (Moyaert et al. 2006). Multi-resistant enterococci are a group of important nosocomial pathogens in human hospitals. Resistance to high-level streptomycin and gentamicin, as in our case, is typical of agents of nosocomial infections in humans, but only very few studies have been published on enterococcal infections in cats and on their antibiotic resistance (Boerlin et al. 2007). *Enterococcus faecium*, isolated from an aerobic culture of liver biopsy after cholecystojejunostomy, exhibited high-level resistance to gentamicin but was susceptible to vancomycin (Jackson et al. 1994). Multidrug-resistant ESBL bacteria, commonly producing CTX-M1 and CTX-M9 group enzymes, have also been isolated from clinical infections in companion animals (Dierikx et al. 2012). Since resistant strains, especially those encoding ESBL enzymes, are being isolated from animals with increasing frequency, antibiotic susceptibility testing has become mandatory in order to ensure successful treatment. While waiting for the results in cases of NF, triple combination antibiotic therapy with penicillin, an aminoglycoside and clindamycin or metronidazole, the latter of which was also used in this case, is recommended for NF in humans (Cunningham et al. 2001). Since NF occurred during the established treatment for suspected viral infection, multiple drug resistance was expected and antibiotic treatment would have been changed if the owner had allowed further treatment. But since treatment requires aggressive surgical debridement, delayed reconstruction (Naidoo et al. 2005) and aggressive supportive care (Jenkins et al. 2001), these were rejected due to the abovementioned financial constraints and the cat was euthanised.

Truncal and perineal infections are thought to have higher mortalities, and delay in diagnosis and surgical management is frequently implicated as an important predictor of increased mortality (Elliott et al. 1996; Cunningham et al. 2001). Unfortunately, the early clinical signs, with erythema, sensitivity and swelling of the area (Cunningham et al. 2001), were here overlooked, and diagnosis was made when the overlying skin had developed ischaemic necrosis. Extensive tissue necrosis, intravascular thrombosis and acute cellular inflammatory response are characteristic histopathological features of NF (Worth et al. 2005), and were also all observed in the present case.

To date, five reports (one of them was a shelter outbreak) of NF have been reported in cats (Brachelente et al. 2007; Pesavento et al. 2007; Sura et al. 2008; Hess 2009; Berube et al. 2010); of these, only one survived (Hess 2009). Some were treated very intensively but nevertheless died.

In retrospect, several aspects of case management could have been carried out differently to have allowed for more effective use of diagnostic testing and treatment. Earlier sampling of the affected area when initial oedema was noted, with incisional biopsy and tissue cultures, would have provided information regarding antimicrobial sensitivity in a more timely fashion. But although we thought from the beginning, that the cat would benefit from intensive care, the owner elected to treat the cat symptomatically and at a very low cost. Under these circumstances, prognosis was poor and the cat was euthanised.

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