Severe acute cellulitis and sepsis caused by *Aeromonas spp.* in a dog on immunosuppressive therapy

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Running title: Severe cellulitis caused by *Aeromonas* in a dog

Abbreviations

ALP - Alkaline phosphatase

ALT - Alanine transferase

CRI - Constant rate infusion

GGT - Glutamyl transpeptidase

IM - Intramuscular

PLT - Platelet

RI = Reference interval

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Abstract

Objective – To describe the clinical presentation, diagnostic investigation and medical management of a dog on immunosuppressive therapy that developed a severe soft tissue infection attributed to *Aeromonas hydrophila/caviae*.

Case summary - A 5-year-old female neutered Border Collie dog was presented for investigation of a rapidly growing skin lesion. The dog had been diagnosed with immune-mediated thrombocytopenia and had been receiving immunosuppressive therapy for 5 weeks. Physical examination at initial presentation revealed no abnormalities except a 6 cm raised, erythematous, firm and painful swelling on the ventral abdomen. Within 12 hours of admission, the lesion had expanded to cover much of the ventrum and some areas had begun to slough. The patient had also become obtunded and exhibited pyrexia, tachypnea, tachycardia as well as extreme pain around the lesion. The dog's clinical signs and hematology results were consistent with sepsis. Histopathology showed severe acute suppurative cellulitis and panniculitis and a heavy growth of *Aeromonas hydrophila/caviae* was obtained on tissue culture. The infection was treated with trimethoprim sulphadiazine, based on culture and susceptibility results.

Unique information provided – This is the first reported case of severe panniculitis and cellulitis caused by *Aeromonas spp.* in a dog. *Aeromonas spp.* should be considered a differential diagnosis for cases of severe soft tissue infection, especially in immune-compromised animals or those with a history of aquatic exposure.

Key words: Severe soft tissue infection, dog, cellulitis, immunocompromised, *Aeromonas*

Introduction

Severe skin and soft tissue infections are commonly reported in human medicine but little has been published in the veterinary literature. *Aeromonas spp* are Gram-negative rods that are ubiquitous in freshwater environments and produce virulent cytotoxins and hemolysins¹ which allow them to initiate opportunistic infections in people and animals.²⁻⁴ Economically important pathogens in aquaculture, *Aeromonas spp.* cause a range of serious aquatic animal diseases such as furunculosis in fish,⁵ septicemia in aquatic reptiles⁶ and necrotic enteritis in birds.⁷ *Aeromonas spp.* are also increasingly important pathogens in people, implicated in 3 main syndromes: gastrointestinal disease in children and adults, skin and soft tissue infections (SSTIs) in healthy and immune-compromised adults and septicemic conditions in immune-compromised and elderly patients.^{2, 8-12} The pathogenicity of *Aeromonas spp* in human medicine and aquaculture is well documented but reports in companion animals are limited to scant reports. One such report documents a fatal leptospirosis-like syndrome in a dog caused by *A. hydrophila*,¹³ while a second report describes *A. hydrophila* septicemia in a litter of 7 puppies.¹⁴ Here we report a case of severe acute panniculitis and cellulitis in a 5-year-old female Border Collie caused by *Aeromonas spp.* Although the current recommended treatment for severe soft tissue infections in both dogs and people includes aggressive surgical debridement,^{15, 16} the patient in this case survived with medical treatment alone.

Case summary

A 5-year-old female neutered Border Collie was presented to our emergency service for investigation of a rapidly growing skin lesion on the ventral abdomen. Five weeks prior to presentation, the dog had been diagnosed with immune-mediated thrombocytopenia which had been treated by the referring veterinarian on an outpatient basis. Treatment had initially included prednisolone^a at 1mg/kg PO q 12h, cyclosporine^b (5mg/kg PO q 12h), omeprazole^c (0.7mg/kg PO q 24h) and sucralfate^d (1g PO q 12h). Azathioprine^e at 1.5mg/kg PO q 24h had been instituted in place of cyclosporine due to failure to respond to therapy. Azathioprine was discontinued 4 days after

starting therapy, due the development of acute pancreatitis diagnosed on the basis of typical clinical signs and an increased canine specific lipase level (655 $\mu\text{g/mL}$, reference interval (RI) $<200\mu\text{g/mL}$). Thoracic radiographs and abdominal ultrasound performed at the time of the diagnosis of pancreatitis had been unremarkable. A mild pustular skin rash was noted on the abdominal skin after the ultrasound and was attributed to irritation from clipping. One week prior to admission to the emergency service, cyclosporine (5mg/kg PO q 12 h) had been restarted when a low platelet count had been noted (platelet (PLT) count= $170 \times 10^9/\text{L}$ ($170 \times 10^3/\mu\text{L}$), RI= $200\text{-}500 \times 10^9/\text{L}$). The dog had been reportedly lethargic, inappetent, polyuric and polydipsic since starting prednisolone therapy, but lethargy had worsened on the day of presentation to the emergency service. The dog was exercised regularly, including swimming in salt water, and was often bathed in water from the town water supply. She was current with vaccinations and parasite control.

On the day of presentation to the emergency service, the dog had also been seen as an outpatient by the referring veterinarian. The presenting complaint to the referring veterinarian was lethargy and the appearance of multiple raised, erythematous cutaneous lesions (2cm diameter) on the ventral abdomen. Serum biochemistry revealed marked increase in alanine transferase (ALT) activity, alkaline phosphatase (ALP) activity and a moderate increase in cholesterol concentration (Table 1). As it was a weekend and the referring veterinarian was closing, the patient was discharged. The dog presented later that day to our emergency service for worsening lethargy and progression of the skin lesions. On presentation to the emergency service, the dog was quiet and responsive with normal vital signs except for a mildly increased respiratory rate (40/min, RI= 10-32/min). The skin lesions had grown over the course of the day to coalesce and form a 6 cm-wide raised, firm and warm erythematous strip along the ventral thorax and abdomen (Figure 1A). The dog was painful on abdominal palpation, especially around the skin lesion. A marker was used to draw the initial margins of the lesion on the patient's skin, to allow tracking of its size. Mild peripheral lymphadenomegaly was noted. Serum biochemistry was performed approximately 20 hours after the initial tests at the referring veterinarian and revealed marked increases in ALT, ALP and gamma glutamyl

transpeptidase (GGT) activities, and moderate increases in lipase activity and cholesterol concentration (Table 1). All other analytes including bilirubin were normal. Hematology revealed hemoconcentration and leukopenia characterized by neutropenia, lymphocytopenia, monocytopenia and mild thrombocytopenia (Table 1). All other hematological values were within RI.

The dog was hospitalized and commenced on isotonic intravenous fluids^f at 4.4mL/kg/hr, buprenorphine hydrochloride^g 0.01mg/kg IM q 6h and esomeprazole sodium^h 0.7mg/kg IV q 24h. Prednisolone was continued at 1mg/kg PO q 12h while cyclosporine was discontinued. After 6 hours in hospital, the dog's rectal temperature had risen to 40.4°C (104.7°F), she was tachycardic (heart rate= 160/min), panting, her mucous membranes were pink and tacky with a capillary refill time of 2 seconds and she appeared alert but uncomfortable. On suspicion of sepsis, a 10 mL jugular venous blood sample was collected using sterile technique and was sent for aerobic blood cultureⁱ prior to commencing intravenous antimicrobials (ticarcillin-clavulanate^j at 50mg/kg IV q 8h). Ticarcillin-clavulanate was chosen as an empirical broad spectrum antimicrobial in accordance with the Infectious Diseases Society of Americas guidelines for using an anti-pseudomonal agent in the management of SSTIs in febrile, neutropenic patients.¹⁷ Increased hemodynamic support was instituted by giving a 10 mL/kg IV bolus of isotonic fluids, then increasing the fluid rate to 6 mL/kg/hr. A urinary catheter was placed to allow monitoring of urine output.

The following morning, the dog was transferred to the hospital's Internal Medicine Service. At the time of transfer the dog was depressed, recumbent and tachypneic (respiratory rate= 128/min), with a heart rate of 160/min, rectal temperature of 41.1°C (106.0°F) and injected mucous membranes. A grade II/VI left apical systolic heart murmur was noted, which had not been noted previously. The skin lesion had progressed along the ventral thorax and abdomen, with extension laterally and dorsally to half-way up the body wall. The erythema had deepened in colour, especially along the mid-ventral abdomen, where a mottled purpuric pattern was evident. There were multiple pustules and small areas of skin were beginning to slough (Figure 1B).

The patient demonstrated extreme pain on gentle palpation of the lesion. To potentiate analgesia, an intravenous CRI of fentanyl^k 5µg/kg/hr and ketamine^l 0.1mg/kg/hr was commenced and buprenorphine was discontinued. As the dog's platelet count was normal and the lesion was extremely painful, necrotizing fasciitis or a severe soft tissue infection were suspected and thought more likely than thrombocytopenic purpura. Other differentials included a cutaneous drug reaction or less likely neoplasia, such as cutaneous hemangiosarcoma. The systolic heart murmur was considered physiological and secondary to pyrexia. Echocardiography to exclude endocarditis and cardiomyopathy was performed by the attending clinician and was unremarkable. To exclude intrathoracic disease as a cause of tachypnea, right lateral and dorsoventral thoracic radiographs were taken and no abnormalities were detected. The patient's oxygen saturation (SpO₂) was normal on room air so tachypnea was attributed to a systemic inflammatory response (SIRS) or sepsis, pain or pyrexia. An ultrasound of the abdominal wall revealed hypoechogenicity between the fascial planes of the ventral abdomen, consistent with severe subcutaneous edema. No free abdominal fluid was evident and the muscular layer of the abdominal wall appeared intact.

Skin biopsies were obtained under gaseous anesthesia^m using an 8 mm skin biopsy punch to provide material for histopathologic assessment and bacterial culture. Extensive debridement was not undertaken at the time, because the subcutaneous tissue did not appear devitalized nor easily dissectible along fascial planes so was not thought to be grossly necrotic.¹⁷ Pending laboratory results, the dog remained in hospital on supportive care (intravenous fluid therapy and fentanyl/ketamine CRI). Ticarcillin-clavulanate (50mg/kg IV q 8h) was continued and metronidazoleⁿ (10mg/kg IV q 12h) was added. Prednisolone^o was also continued at 1mg/kg IV q 24h as acute discontinuation of prednisolone may have precipitated a hypoadrenocorticoid crisis. Over the next 48 hours, the dog responded well to supportive therapy with an improved demeanor and return of appetite, although fluctuating pyrexia and tachypnea remained. On day 4 of hospitalization, the dog's platelet count dropped to 70 x 10⁹/L (70x10³/µL, RI= 200-500x10⁹/L) so the dose of prednisolone was increased to 1mg/kg PO q 12h. Urine production remained adequate (1 mL to 3.8 mL/kg/hr). Fentanyl and

ketamine were weaned after 4 days and analgesia was switched to tramadol hydrochloride^p (2mg/kg PO q 8h). The ventral abdominal skin lesion remained erythematous and edematous and the central area became necrotic and ultimately sloughed to form a 7x5cm open wound (Figure 1C).

Histopathology demonstrated marked acute extensive suppurative panniculitis and cellulitis with intralesional bacteria. Although necrosis was noted, the predominant process was considered to be more suppurative than necrotic. The aerobic blood culture yielded no bacterial growth after 5 days. A heavy growth of *Aeromonas hydrophila/caviae* was cultured from the fresh biopsy samples. The aeromonad isolate was not subspeciated beyond *A. hydrophila/caviae*. The isolate was susceptible to trimethoprim sulphadiazine, gentamicin and cefovecin and was resistant to amoxicillin-clavulanic acid and cephalixin. It was not specifically tested against ticarcillin-clavulanate. Antimicrobial therapy was accordingly altered to trimethoprim sulfadiazine^q (15mg/kg PO q 12h). Pyrexia resolved with the change of antimicrobials and the skin lesion also began to resolve. Consequently, the dog was deemed well enough to be treated as an outpatient but remained at the hospital at the owner's request, for ease of wound management. The wound on the ventral abdomen was managed with a wet to dry bandage with daily gentle debridement.

After 14 days in hospital, debridement was complete and the wound had begun to heal by second intention. CBC showed the dog's platelet count had normalized (Table 1). The remaining deficit was closed through delayed primary closure under inhalational anesthesia.^m The dog was discharged on day 14 on trimethoprim sulphadiazine (15mg/kg PO q 12h), metronidazole^r 10mg/kg PO q 12h and prednisolone 1mg/kg PO q 12h. The dog returned every 2 weeks for clinical reassessment, where prednisolone was tapered and eventually discontinued after 4 months. All hematology and serum biochemistry values had returned to normal at the 4 month (day 128) revisit (Table 1). Trimethoprim sulfadiazine and metronidazole were discontinued after 6 weeks. Four years after clinical resolution of the skin lesion, the dog is receiving no medications and is still clinically well with no recurrence of immune-mediated thrombocytopenia or skin lesions.

Discussion

Aeromonas spp. are important causes of skin and soft tissue infections in people^{8, 10, 11} and systemic *Aeromonas* infections have been reported in dogs,^{13, 14} but this is the first time that a severe soft tissue infection attributable to *Aeromonas* spp. has been reported in a dog. The isolate was not speciated beyond *A. hydrophila* or *A. caviae*, but both are plausible pathogens. *A. hydrophila* is a more common cause of SSTIs in people than *A. caviae*,¹⁰ and *A. hydrophila* exhibits more virulence factors ex vivo than *A. caviae*.¹⁸ However, both species have been retrieved from rectal swabs of dogs¹⁹ and have been isolated from severe soft tissue infections in people,^{8, 10, 11} so either agent could have created disease in this case.

The source of *Aeromonas* spp. is not known in this case, but it is likely that aquatic exposure and immunosuppressive therapy played roles in this opportunistic infection. The dog's history of regular swimming and recent 'clipper rash' echoes the literature, where historical findings of skin trauma and aquatic exposure are common amongst *Aeromonas* spp. SSTIs in people¹⁰ and in the case of fatal *A. hydrophila* infection reported in a dog.¹³ *Aeromonas* spp. are ubiquitous in aquatic environments,³ which would explain why aquatic exposure is a common finding in *Aeromonas* SSTIs^{8, 10, 12, 13} and why *Aeromonas* is a significant aquatic animal pathogen.^{5, 6} Besides aquatic exposure, *Aeromonas* spp. are also shed in the feces of some dogs¹⁹ so it is also possible that the infection was endogenous. The dog's history of immunosuppressive therapy is consistent with cases of SSTIs in the veterinary literature¹⁵ but is an inconsistent historical finding amongst *Aeromonas*-associated SSTIs in people. Most dogs that develop bacterial SSTIs have an underlying disease that affects their immune function or skin integrity.¹⁵ In contrast, *Aeromonas*-associated SSTIs are often reported in healthy human patients with no evidence of immune-compromise.^{8, 10, 12, 13, 20} Consequently, while the dog in this particular case was immunocompromised, *Aeromonas* spp. SSTIs should be considered a differential for SSTI in any animal with a history of aquatic exposure.

The dog's clinical signs of pyrexia and a swollen, painful, erythematous and suppurative skin lesion pyrexia are also all consistent with reports of cellulitis caused by *Aeromonas* spp. in the human literature.^{8, 12} The dog's presenting complaint of severe pain is in contrast to SSTIs caused by other bacteria in dogs, where a case series noted pain in only 6 of 47 dogs with SSTIs.¹⁵ The authors of that study conceded that pain may have not been recognized in severely obtunded patients, so the prevalence of pain may have been underestimated.¹⁵ Pain that is out of proportion to clinical findings is also an indicator for a necrotizing process²¹ and hence necrotizing fasciitis was on the differential list. Although necrotizing fasciitis was not present on histopathology, extreme pain exhibited in this case have been elicited from substantial tissue damage and inflammation caused by the *Aeromonas* cytotoxins.^{1, 22}

Aeromonas septicemia is a well-recognized condition in people and although *Aeromonas* was not isolated from the single blood culture performed in this case, the dog's tachycardia, tachypnea, leucopenia, and isolation of a Gram-negative organism all fulfilled the diagnostic criteria for SIRS and Gram-negative sepsis.^{23, 24} Although rare, *Aeromonas*-associated sepsis is well documented in immunocompromised people.² In this case, leucopenia was initially attributed to immunosuppressive therapy, but as the dog deteriorated, sepsis was thought a more likely cause. Similarly, while prednisolone therapy could have caused increases in the ALP activity, we observed marked increases in ALT, ALP and GGT activities. Such increases are consistent with hepatobiliary abnormalities that are described in other *Aeromonas* cases.^{13, 14} *A. hydrophila* was isolated from the livers of a litter of puppies who died of *A. hydrophila* infection¹⁴ and from the liver of a dog with congestive hepatitis who died of a presumed septicemic *A. hydrophila* infection.¹³ Pre-existing hepatobiliary disease is one of the common predisposing conditions for *Aeromonas* infections in people,^{25, 26} but this patient had no evidence of prior history of liver dysfunction. Additionally, the dog's liver enzyme activities had returned to normal at its 4-month recheck so it did not appear to have a primary liver condition. Therefore, we feel that most of the hemodynamic and biochemical changes in this case can be explained by the effects of sepsis.

While the dog's sepsis was recognized and addressed with medical management, early and aggressive surgical debridement was not undertaken and this is in contravention to what is generally recommended for severe soft tissue infections in both people¹⁶ and animals.¹⁵ Extensive debridement was not undertaken initially because the dog's lesions appeared more inflammatory than necrotic, an assumption that was confirmed by histopathology of the biopsy specimen. Had necrosis been present, aggressive surgical debridement would likely have been required to obtain clinical resolution,^{15, 16, 21} although a recent case series described 3 cats with necrotizing fasciitis that survived without surgical debridement.²⁷ When the dog began to improve with medical therapy, it was decided that debridement would not significantly improve the dog's clinical outcome. Nevertheless, the fact that some of the skin lesion eventually sloughed shows that the tissue became devitalized and we concede that debridement may well have shortened the clinical course of disease.

Part of the medical management of this case involved empirical therapy with a broad spectrum antimicrobial, ticarcillin-clavulanate. β -hemolytic *Streptococcus* spp. and *Pseudomonas aeruginosa* are both recognized causes of severe soft tissue infections in dogs^{15, 28} so ticarcillin-clavulanate was a reasonable empirical choice, although ampicillin would have been a superior choice for a β -*Streptococcus* spp. SSTI.²⁹ However, *Aeromonas* spp. are almost universally resistant to ampicillin,^{8, 13} so it would not have helped in this case. The addition of metronidazole was most likely unnecessary, as any anaerobic pathogens would likely have been susceptible to ticarcillin-clavulanate.³⁰ Although not tested by culture and susceptibility, it is likely the *Aeromonas* isolate in this case was clinically resistant to ticarcillin-clavulanate,³¹ evidenced by continuation of the dog's pyrexia while on ticarcillin-clavulanate and prompt cessation of the dog's pyrexia once therapy was switched to trimethoprim sulphadiazine. Failure of empirical therapy in this case is not surprising, as empirical antimicrobial therapy often fails to provide coverage of *Aeromonas* spp. infections in people.⁸ Antimicrobial therapy could have been de-escalated and metronidazole discontinued once culture and susceptibility results revealed the *Aeromonas* isolate was susceptible to trimethoprim

sulphadiazine.³² Early antimicrobial choice could have been improved in this case by examining the cytology and gram stain of a fine needle aspirate of the initial lesion in-house.

In conclusion, *Aeromonas spp.* should be included in the differential diagnosis for severe soft tissue infections in dogs, particularly in immune-compromised patients that have been exposed to aquatic environments. Although the *Aeromonas* isolate in this case was susceptible to several antimicrobials, veterinarians should be aware of the public health risk posed by *Aeromonas spp.*, given their tendency for intrinsic antimicrobial resistance and their demonstrated potential as a fatal human and animal pathogen.

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Footnotes

^a Pred-X, Apex Laboratories Pty Ltd, Somersby, Australia

^b Atopica, Novartis Animal Health, Basel, Switzerland

^c Losec, AstraZeneca Pty Ltd, North Ryde, Australia

^d Carafate, Aspen Pharmacare Pty Ltd, St Leonards, Australia

^e Imuran, Aspen Pharmacare Pty Ltd, St Leonards, Australia

^f Hartmann's Solution, Baxter Health Care Pty Ltd, Old Toongabbie, Australia

^g Temgesic, Reckitt Benckiser, West Ryde, Australia

^h Nexium, AstraZeneca Pty Ltd, North Ryde, Australia

ⁱ Oxoid Signal blood culture system, Oxoid Thermo Scientific, Basingstoke, United Kingdom

^j Timentin, GlaxoSmithKline Australia Pty Ltd, Abbotsford, Australia

^k DBL fentanyl citrate, Hameln Pharmaceuticals GmbH, Langes Field, Germany

^l Ketamine hydrochloride, Parnell Australia Pty Ltd, Alexandria, Australia

^m Delvet Isoflurane Inhalation Anaesthetic, Ceva Delvet Pty Ltd, Seven Hill, Australia

ⁿ Metronidazole, Baxter Laboratories, Old Toongabbie, Australia

^o Solu-delta-cortef, Pfizer Pty Ltd Australia, West Ryde, Australia

^p Tramadol hydrochloride, Apex Laboratories Pty Ltd, Somersby, Australia

^q Tribissen 80, Jurox Pty Ltd, Rutherford, Australia

^r Metrogyl, Alphapharm Pty Ltd, Carole Park, Australia

References

1. Ghatak S, Agarwal R, Bhilegaonkar K. Comparative study of cytotoxicity of *Aeromonas* spp. on four different cell lines. *Comp Immunol Microbiol Infect Dis*. 2006;29(4):233-41.
2. Janda JM, Abbott SL. The genus *Aeromonas*: taxonomy, pathogenicity, and infection. *Clin Microbiol Rev*. 2010;23(1):35-73.
3. Janda JM, Bottone EJ, Reitano M. *Aeromonas* species in clinical microbiology: significance, epidemiology, and speciation. *Diagn Microbiol Infect Dis*. 1983;1(3):221-8.
4. Voss LM, Rhodes KH, Johnson KA, editors. Musculoskeletal and soft tissue *Aeromonas* infection: an environmental disease. *Mayo Clin Proc*; 1992;67(5): 422-427.
5. McCarthy D. An experimental model for fish furunculosis caused by *Aeromonas salmonicida*. *J Fish Dis*. 1983;6(3):231-7.
6. Shotts E, Gaines J, Martin L, Prestwood AK. *Aeromonas*-induced deaths among fish and reptiles in an eutrophic inland lake. *J Am Vet Med Assoc*. 1972;161(6):603-7.

7. França M, Walker R, Kokka R, Shivaprasad H. *Aeromonas* species associated with necrotizing enteritis and septicemia in an adult male ostrich (*Struthio camelus*). Avian Dis. 2009;53(2):310-6.
8. Gold W, Salit I. *Aeromonas Hydrophila* infections of skin and soft tissue: report of 11 cases and review. Clin Infect Dis. 1993;16(1):69-74.
9. Clark NM, Chenoweth CE. *Aeromonas* infection of the hepatobiliary system: report of 15 cases and review of the literature. Clin Infect Dis. 2003;37(4):506-13.
10. Chao C, Lai C, Tang H, Ko W, Hsueh P-R. Skin and soft-tissue infections caused by *Aeromonas* species. Eur J Clin Microbiol Infect Dis. 2013;32(4):543-7.
11. Kumar S, Mukhopadhyay P, Chatterjee M, et al. Necrotizing fasciitis caused by *Aeromonas Caviae*. Avicenna J Med. 2012;2(4):94-6.
12. Kelly KA, Koehler JM, Ashdown LR. Spectrum of Extraintestinal Disease Due to *Aeromonas* Species in Tropical Queensland, Australia. Clin Infect Dis. 1993;16(4):574-9.
13. Andre-Fontaine G, Monfort P, Buggin-Daubie M, et al. Fatal disease mimicking leptospirosis in a dog, caused by *Aeromonas Hydrophila*. Comp Immunol Microbiol Infect Dis. 1995;18(1):69-72.
14. Zdovec I, Švara T, Juntos P, et al. The role of *Aeromonas Hydrophila* bacterium as a causative agent of septicemia in dogs. Slov Vet Res. 2004; 41 (1):23-30.
15. Buriko Y, Van Winkle TJ, Drobatz KJ, Rankin SC, Syring RS. Severe soft tissue infections in dogs: 47 cases (1996–2006). J Vet Emerg Crit Care. 2008;18(6):608-18.
16. Bilton BD, Zibari GB, McMillan RW, Aultman DF. Aggressive surgical management of necrotizing fasciitis serves to decrease mortality: a retrospective study/discussion. Am Surg. 1998;64(5):397-400.
17. Stevens DL, Bisno AL, Chambers HF et al. Practice Guidelines for the Diagnosis and Management of Skin and Soft Tissue Infections: 2014 Update by the Infectious Diseases Society of America. Clin Infect Dis. 2014;59(2):e10-e52.

18. Chen P-L, Wu C-J, Tsai P-J et al. Virulence diversity among bacteremic *Aeromonas* isolates: ex vivo, animal, and clinical evidences. PLoS One. 2014;9(11):e111213.
19. Ghenghesh KS, Abeid SS, Jaber MM, Ben-Taher SA. Isolation and haemolytic activity of *Aeromonas* species from domestic dogs and cats. Comp Immunol Microbiol Infect Dis. 1999;22(3):175-9.
20. Hassan V, Amanda W, Cameron S, Gary KD, Tony W. Outbreak of *Aeromonas hydrophila* Wound Infections Associated with Mud Football. Clin Infect Dis. 2004;38(8):1084-9.
21. Rogers RL, Perkins J. Skin and soft tissue infections. Prim Care. 2006;33(3):697-710.
22. Parker JL, Shaw JG. *Aeromonas* spp. clinical microbiology and disease. J Infect. 2011;62(2):109-18.
23. Otto CM. Sepsis in veterinary patients: what do we know and where can we go? J Vet Emerg Crit Care. 2007;17(4):329-32.
24. Hauptman JG, Walshaw R, Olivier NB. Evaluation of the Sensitivity and Specificity of Diagnostic Criteria for Sepsis in Dogs. Vet Surg. 1997;26(5):393-7.
25. DeFronzo RA, Murray GF, Maddrey WC. *Aeromonas* septicemia from hepatobiliary disease. The American journal of digestive diseases. 1973;18(4):323-31.
26. Duthie R, Ling T, Cheng A, French G. *Aeromonas* septicaemia in Hong Kong species distribution and associated disease. J Infect. 1995;30(3):241-4.
27. Vaske HH, Ragan IK, Harkin KR, Lee K, Hicks SE. Successful conservative management of suspected Fournier's gangrene in cats: three cases. J Fel Med Surg Open Reports 2015;1(1):1-9.
28. Hillier A, Alcorn JR, Cole LK, Kowalski JJ. Pyoderma caused by *Pseudomonas aeruginosa* infection in dogs: 20 cases. Vet Dermatol. 2006;17(6):432-9.
29. Pfaller MA, Jones RN. In vitro evaluation of contemporary β -lactam drugs tested against viridans group and β -haemolytic streptococci. Diagn Microbiol Infect Dis. 1997;27(4):151-4.
30. Citron DM, Goldstein EJC, Kenner MA, Burnham LB, Inderlied CB. Activity of Ampicillin/Sulbactam, Ticarcillin/Clavulanate, Clarithromycin, and Eleven Other Antimicrobial

Agents Against Anaerobic Bacteria Isolated from Infections in Children. Clin Infect Dis.

1995;20(Supplement_2):S356-S60.

31. Vila J, Marco F, Soler L, Chacon M, Figueras MJ. In vitro antimicrobial susceptibility of clinical isolates of *Aeromonas caviae*, *Aeromonas hydrophila* and *Aeromonas veronii* biotype *sobria*.

J Antimicrob Chemother. 2002;49(4):701-2.

32. Rhodes A, Evans LE, Alhazzani W et al. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock: 2016. Intensive Care Med. 2017;43(3):304-

77.

Table 1. Summary of clinicopathological findings from a dog on immune-suppressive therapy with severe acute *Aeromonas* cellulitis and panniculitis. Values in bold indicate abnormal results that fall outside the reference interval.

Analyte	SI units (conventional)	Day 1	Day 2	Day 4*	Day 14	Day 128	Reference interval
ALT	U/L	908	897	-	-	65	10-100
ALP	U/L	1246	3044	-	-	32	23-212
GGT	U/L	ND	351	-	-	-	10-70
Lipase	U/L	ND	2041	-	-	511	200-1800
Cholesterol	mmol/L	10.9	10.73	-	-	7.2	2.34-8.26
	(mg/dL)	(436)	(429)			(288)	(110-320)
HCT	L/L (%)	0.60	0.42	0.33	0.30	0.48	0.35-0.55
		(60%)	(42%)	(33%)	(33%)	(48%)	(35-55%)
WBC	x10 ⁹ /L	-	4.43	2.6	19.1	9.1	5.5-16.9
	(x10 ³ /μL)						
Neutrophils	x10 ⁹ /L	-	2.9	1.7	13.8	6.5	3.5-12.0
	(x10 ³ /μL)						
Lymphocytes	x10 ⁹ /L	-	0.1	0.3	2.5	1.9	0.9-3.5
	(x10 ³ /μL)						

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Monocytes	x10 ⁹ /L	-	0.2	0.4	1.7	0.5	0.3-2.0
	(x10 ³ /μL)						
Platelets	x10 ⁹ /L	170	191	70	636	123**	200-500
	(x10 ³ /μL)						
ALT= alanine transferase, ALP= alkaline phosphatase, GGT= Gamma glutamyl transpeptidase, HCT= hematocrit, WBC= white blood cells. * = On day 4, liver enzymes were measured and recorded as 'stable' but unfortunately the results were discarded. ** = On day 128, platelets were clumped and adequate on a manual blood smear							

Figure 1. Rapidly progressive erythematous skin lesion caused by *Aeromonas* spp. cellulitis and panniculitis on the ventral abdomen of a 5-year-old female spayed Border Collie dog. A= day 1, B= day 2, C= day 8

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