NOTE
Internal Medicine

Pancreatic Abscess in a cat due to *Staphylococcus aureus* infection

Yuki NEMOTO[1]*, **Tomoya HARAGUCHI**[2], **Takako SHIMOKAWA MIYAMA**[3], **Kosuke KOBAYASHI**[3], **Kaori HAMA**[3], **Yosuke KUROGOUCHI**[1], **Noriyuki FUJIKI**[1], **Kenji BABA**[3], **Masaru OKUDA**[3] and **Takuya MIZUNO**[1]

[1] Laboratory of Molecular Diagnostics and Therapeutics, Department of Veterinary Medicine, Joint Faculty of Veterinary Medicine, Yamaguchi University, 1677-1 Yoshida, Yamaguchi 753-8515, Japan
[2] Laboratory of Veterinary Surgery, Department of Veterinary Medicine, Joint Faculty of Veterinary Medicine, Yamaguchi University, 1677-1 Yoshida, Yamaguchi 753-8515, Japan
[3] Laboratory of Veterinary Internal Medicine, Joint Faculty of Veterinary Medicine, Yamaguchi University, 1677-1 Yoshida, Yamaguchi 753-8515, Japan

ABSTRACT. A 16-year-old spayed female American Shorthair cat was presented with lethargy, anorexia, and wamble. Physical and blood examination did not reveal any remarkable findings. Abdominal ultrasonography identified the presence of a localized anechoic structure with a thick wall in contact with the small intestine and adjacent to the liver. Ultrasound-guided fine-needle aspiration of the structure revealed fluid containing numerous cocci and neutrophils. Two days after antibiotic treatment, exploratory laparotomy was performed and the content of the structure was removed before multiple lavages. The pathological and bacteriological examination results supported a confirmatory diagnosis of pancreatic abscess due to *Staphylococcus aureus* infection, making this the first such report in a cat. The cat remained healthy thereafter with no disease recurrence.

KEY WORDS: cat, pancreatic abscess, pancreatitis, *Staphylococcus aureus*

Pancreatic masses such as pancreatic pseudocysts, necrotic mass lesions, and pancreatic abscesses are uncommon in cats and dogs [1]. Pancreatic pseudocyst, which usually develops secondary to pancreatitis, is a non-epithelialized sac formed by fibrous and granulation tissue containing fluid made up of pancreatic enzyme and debris [4, 12]. On the other hand, pancreatic necrosis is defined as a diffuse or focal area of non-viable pancreatic parenchyma, typically associated with peripancreatic fat necrosis [6]. Found as a collection of septic exudate, pancreatic abscess is due to secondary infection of necrotic pancreatic tissue or pancreatic pseudocyst [6]. In human medicine, pancreatic abscess has also been defined as a lesion with purulent material, positive in bacteriological culture, but lacking of necrosis, which emphasizes the differences with pancreatic necrosis [6]. Due to the infrequent report of pancreatic masses in veterinary medicine, the definition of pancreatic abscess has remained ambiguous in animals. Sterile bacteriological cultures of pancreatic abscesses in dogs have been reported [1, 7, 12], while pancreatic abscess in cats is almost unheard of. Based on the currently available information, the isolation of microbial agents is much less frequent in veterinary medicine.

In this report, a case of a cat with pancreatic abscess arising from *Staphylococcus aureus* (*S. aureus*) infection was successfully treated with a combination of antibiotics, surgical removal of purulent material from the abscess, and multiple lavages.

A 16-year-old spayed female American Shorthair cat, weighing 2.8 kg, was referred to the Yamaguchi University Animal Medical Center (Day 1) with clinical signs of lethargy, anorexia, and wamble, which had started 5 days before admission. She was the only pet at home and was kept indoors. Upon physical examination, she appeared to be normal but with poor body condition (a score of 2 out of 9). Parameters of the complete blood cell count (CBC) and blood biochemistry examinations were within normal limits, except for elevated segmented neutrophils ($395.28 \times 10^2/\mu l$; reference range, $40–150 \times 10^2/\mu l$), and blood glucose (214 mg/d*l*; reference range, 71–148 mg/d*l*). The concentration of serum 3,5,3′5′-tetraidothyronine (T_4), free T_4, and thyrotropin (TSH) were also within normal limits. Results of feline immunodeficiency virus (FIV) antibody/feline leukemia virus (FeLV) antigen test kits (SNAP FIV/FeLV comb, IDEXX Laboratories, Tokyo, Japan) were negative. Urinalysis results were unremarkable and urine glucose non-detectable.

Upon further examination, abdominal ultrasonography revealed a localized anechoic structure with a thick wall, which seemed to be in contact with the small intestine and adjacent to the liver (Fig. 1 A). The structure was approximately 21 × 33 mm and

---

*Correspondence to: Nemoto, Y., Laboratory of Molecular Diagnostics and Therapeutics, Department of Veterinary Medicine, Joint Faculty of Veterinary Medicine, Yamaguchi University, 1677-1 Yoshida, Yamaguchi 753-8515, Japan. e-mail: y.nemoto@yamaguchi-u.ac.jp
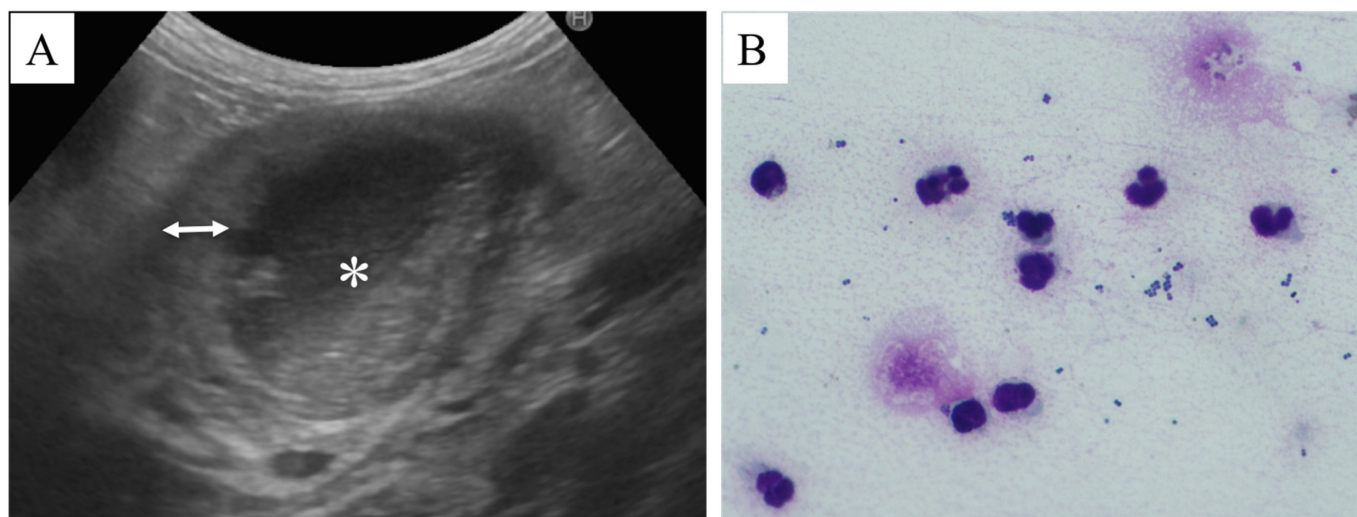
Fig. 1. Abdominal ultrasonographic image showing a localized anechoic structure (*) with a thick wall (↔) in contact with the small intestine and adjacent to the liver. (A) Image from the cytological examination of the fluid inside the structure indicating the presence of numerous cocci and neutrophils. Wright-Giemsa staining, original magnification ×1,000. (B)

appeared to be fluid-filled with a lack of blood flow as seen through color Doppler ultrasonography. This structure was not detectable in the abdominal radiography. Ultrasound-guided fine-needle aspiration (FNA) was performed and about 5 m*l* of a pale pink fluid were aspirated. Cytological examination revealed that the fluid contained numerous cocci and neutrophils (Fig. 1 B). The fluid aspirated from the structure, together with a blood sample, was sent for bacteriological culture. Even though the results of blood examination and urinalysis did not show remarkable abnormalities, the most likely differential diagnoses at that point of time were intestinal neoplasia and abscess. Based on these differential diagnoses, the cat was hospitalized and scheduled for an exploratory laparotomy on Day 3, which was the earliest time for our hospital. Upon hospitalization, the cat was given Ringer's solution (Veen F, Kowa Co., Aichi, Japan) and 20 mg/kg of ampicillin (Amipenix, Kyoritsu Seiyaku, Tokyo, Japan) TID intravenously, while 5 mg/kg of enrofloxacin (Baytril, Bayer, Osaka, Japan) was given SID subcutaneously. The cat's condition improved on the next day and she began to show interest in food. Blood examination was performed again on Day 3 (day of surgery) as a part of pre-surgical monitoring. The results were similar to the results of the first blood examination where elevated segmented neutrophils ($211.1 \times 10^2/\mu l$) and blood glucose (154 mg/d*l*) persisted. The other blood parameters were within normal limits. Computed tomography (CT) was performed pre-surgically to determine the origin of the fluid-filled structure and to see if extirpation of the structure was possible. Based on the CT image, the structure appeared to be originating either from the small intestine or the pancreas, and seemed to be enclosing the common bile duct, which would make the removal of the whole structure difficult (Fig. 2). During exploratory laparotomy, the structure was found to be adhering to the common bile duct as seen in the CT image, and was in contact with the small intestine without being invasive. The structure appeared to have originated from the right lobe of the pancreas and another small mass with thick wall was also found attached to the structure (Fig. 3A). As expected, the entire structure could not be excised due to adhesion to the common bile duct. Therefore, the structure was incised and approximately 6.5 m*l* of fluid containing small hard solids were removed using a syringe before multiple lavages, while taking precautions to avoid spillage into the abdominal cavity. The structure and its immediate surrounding were thoroughly checked and there was no adhesion to the small intestine or any other organ. An additional incision was made on the other side of the structure and omentalization was performed by placing a part of the omentum through the wall of the structure at the end of the surgery (Fig. 3B). The abdominal cavity was closed up using standard procedure and an esophagostomy tube was placed in order to fulfill the nutritional needs of the cat.

Samples from the abnormal structure, including the wall with the attached pancreas, the small hard solids in the structure fluid, and the attached mass were examined for pathological diagnosis. The fluid inside the structure contained cocci similar to the fluid aspirated via ultrasound during the initial examination, and it was examined for bacterial identification. The biopsy of the bile duct and small intestine were not performed in this operation due to the body condition of the cat. Since the structure was considered to be originated from the pancreas based on the intraoperative observation, a frozen blood sample from the initial examination was examined for the Spec fPL test by IDEXX laboratories [17]. The structure's fluid was not evaluated for the concentration of amylase and lipase.

The bacteriological culture results of the blood and the fluid aspirated from the pancreatic abscess via ultrasound-guided FNA revealed the presence of only one type of bacteria, namely *S. aureus*. Based on the intraoperative appearance and the culture result, the confirmatory diagnosis was pancreatic abscess. Based on the results of the antibiotic sensitive test, ampicillin was replaced with 25 mg/kg of cefazolin (Cefamezinα, Astellas, Tokyo, Japan), while enrofloxacin was continued at the same dosage. Camostat mesilate (Foipan, Ono pharmaceutical, Osaka, Japan) was also added. On Day 5 (2 days post-surgery), the cat's appetite improved
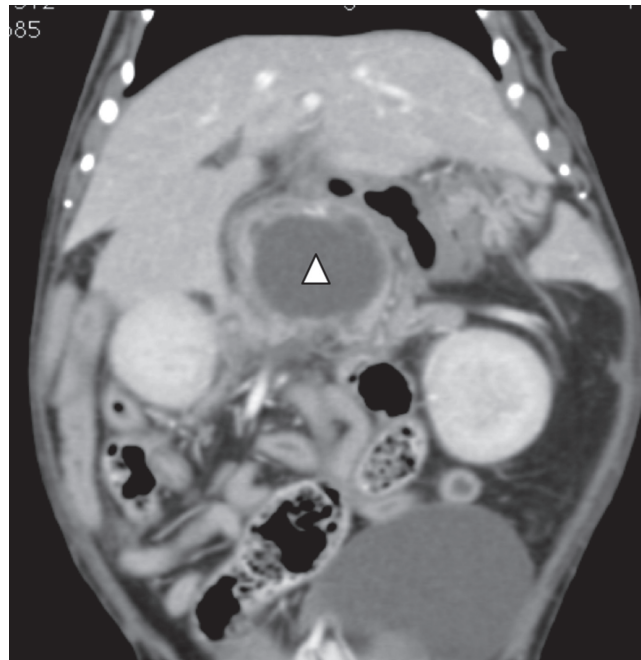
Fig. 2. Computed Tomography (CT) image where the structure (Δ) appeared to be originating either from the small intestine or the pancreas and seemed to be enclosing the common bile duct.

and the esophagostomy tube was maintained only for medication purposes. Blood biochemistry and CBC examinations were performed on Days 4, 5 and 7 and elevated segmented neutrophils (331.5, 325.1 and 254.2 [$\times 10^2/\mu l$], respectively) and blood glucose (149, 148 and 163 mg/d*l*, respectively). The other blood examination parameters were within normal limits. Due to the owner's request, the cat was discharged on Day 8 (5 days post-surgery). Antibiotics lasting for 1 week were prescribed and the owner was advised to bring the cat for a re-examination a week later.

Pathological examination revealed that the structure was chronic active pancreatitis, panniculitis, and fibrosis (Fig. 4). The wall of the pancreatic abscess with attached pancreas consisted of fat tissue, pancreatic tissue, and a part of the mesothelium. Moreover, there was a severe purulent and fibrinous inflammation. The central part of the inflammation consisted of eosinophilic foreign substances, which were possibly bile or gastrointestinal content. As for the small hard solids in the fluid, they were made up of fragments of pancreatic tissue, fibrin, denatured neutrophils, and red blood cells. The mass attached to the pancreatic abscess comprised the same component as the wall of the abscess. None of the samples sent for pathological examination came back with findings related to tumor or microbiological agents. The bacteriological culture results of the fluid collected directly from the inside of the pancreatic abscess during surgery came back positive for *S. aureus*, which was a match with the sample collected via ultrasound-guided FNA. The result of the Spec fPL test performed using the sample from the initial examination was 34.5 μg/*l* (reference range <3.6 μg/*l*). As for the results of histopathological examination and bacterial identification, the samples of the structure with pancreas revealed pancreatitis and pancreatic abscess with *S. aureus*.

The pancreatic abscess was monitored until Day 98. The esophagostomy tube was removed on Day 14 because the cat's appetite returned to an acceptable level. The result of Spec fPL test returned within normal limits on Day 21 (2.9 μg/*l*). The fluid in the pancreatic abscess was checked using ultrasound-guided FNA and was normal, containing denatured neutrophils, but no bacteria. On Day 30, the fluid was found to contain primarily of eosinophil. Therefore, in additional to the antibiotic, 0.8 mg/kg of prednisolone was prescribed for a week. Starting from Day 55, since the fluid did not contain any eosinophil and only neutrophils were seen, all medications were stopped. According to the examination on Day 98, the abscess resolved completely without disease recurrence.

The characteristics of pancreatic abscesses have been reported to be different among species such as humans, dogs, and cats. Both in humans and dogs, pancreatic abscesses are considered to be a late complication of pancreatitis; however, unlike in humans, most pancreatic abscesses in dogs are sterile [2, 6]. As for cats, the characteristics of pancreatic abscesses are considered to be totally different from dogs. To the best of our knowledge, there are five reports related to pancreatic masses in cats, comprising pancreatic pseudocysts, necrosis, and abscesses [9, 11, 12]. Among those, only two cases were reported to have pancreatic abscesses with positive bacteriological culture results, and the causative microbiological agent was *Escherichia coli* in both cases [9, 11]. Here, we anticipated that inflammation of the pancreas may have promoted colonic bacterial translocation. This has been supported by studies of experimental feline pancreatitis; where they have clearly demonstrated that *Escherichia coli* can translocate from the intestines into the acutely inflamed pancreas [14–16].

In our report, *S. aureus* was detected both in the pancreatic abscess and the blood of the cat. *S. aureus* has been reported to
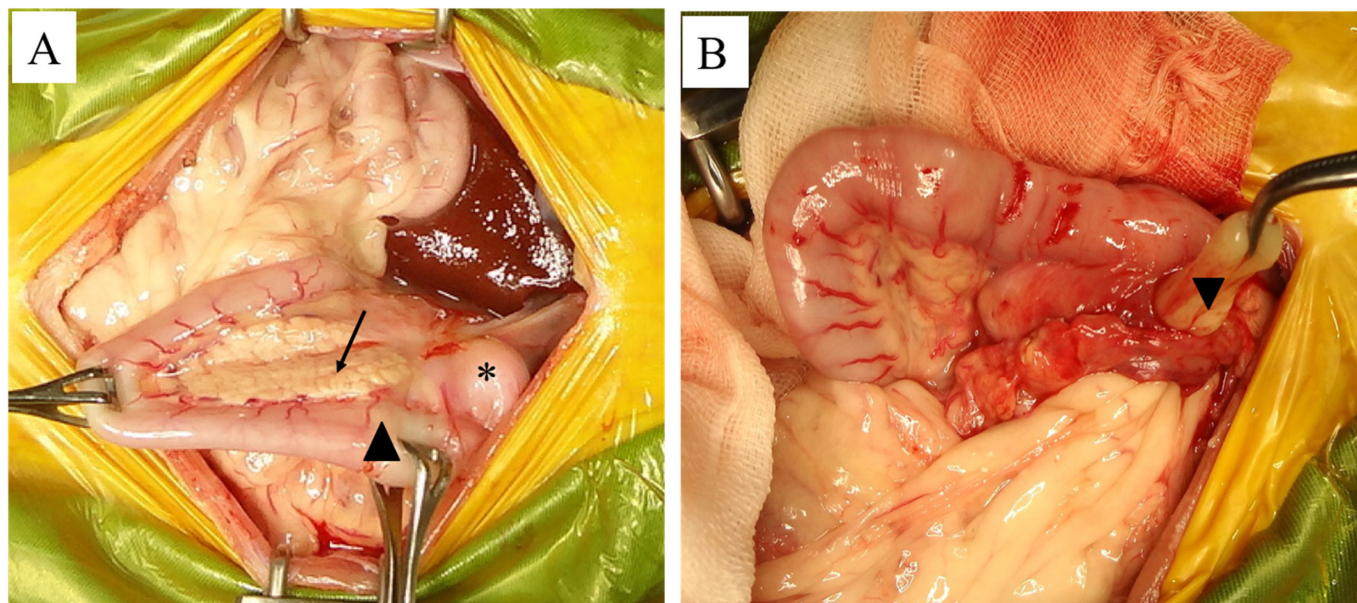
Fig. 3. Intraoperative images (cranial on the left and caudal on the right) indicating that the structure (*) originated from the right pancreatic lobe (→) and it was in contact with the small intestine (▲). (A) The omentum (▼) was placed through the wall of the structure at the end of the surgery (B).
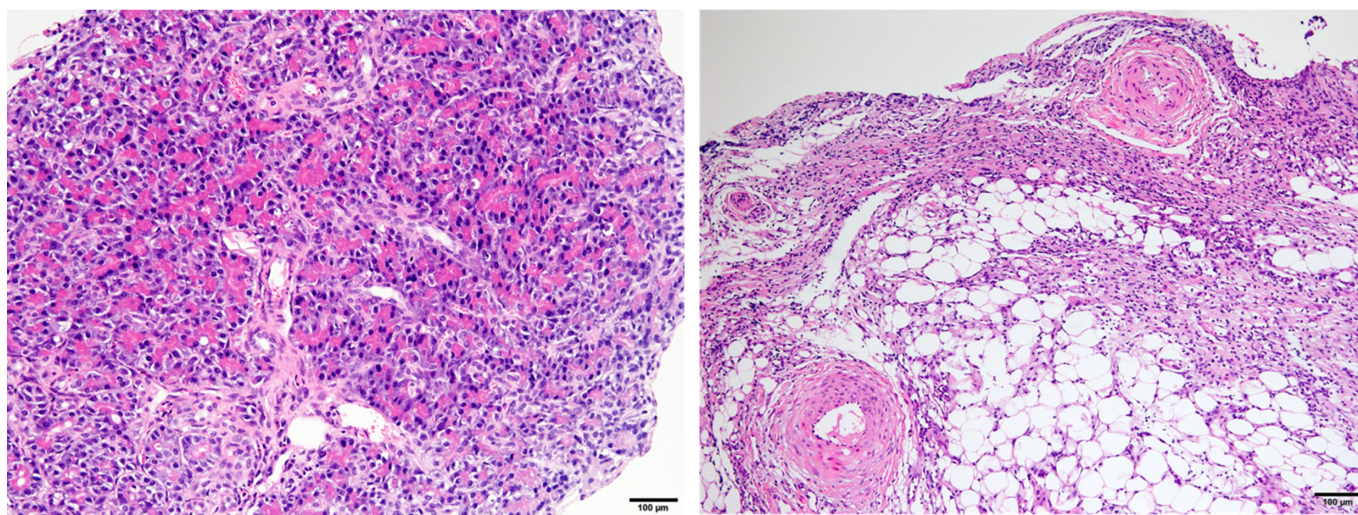

Fig. 4. Histopathology of the mass attached to the pancreatic abscess (HE); chronic active pancreatitis, panniculitis, and fibrosis.

be one of the commensal bacterium as part of the skin and mucosal microflora of the cat [10]. Two possible origins of bacterial infection could be described in this cat, namely the colon and the skin. As the pathological examination showed that the abscess contained substances from the bile and small intestine, we suggest that bile and intestinal bacterial translocation could have taken place through inflammation of the pancreas, which is consistent with the reports on *Escherichia coli* and other enteric bacteria previously described [9, 11]. In humans, *S. aureus* in pus disseminates onto skin surfaces or enters the circulating lymph, and blood sepsis often disseminates to form abscesses at distal sites and affect virtually any internal organ system [5, 8]. Although *S. aureus* was found on the skin of this cat, the origin of the pancreatic abscess was not clear since the owner was not eager to have more examinations regarding the skin disease. In humans, *S. aureus* onto skin might form the abscess on the pancreas. Although there are no reports on this in cats, we suggest that, similarly to humans, *S. aureus* may also tend to form pancreatic abscesses in cats. More cases will be needed for comparison in order to investigate and obtain additional information about pancreatic abscesses in cats.

The most important treatment of abscesses is the removal of the purulent material. In human medicine, once pancreatic abscess is diagnosed, drainage is considered to be essential. Moreover, open abdominal surgical intervention and endoscopic surgery are performed because an infected necrotic region without surgical intervention is fatal [2, 3, 13]. In veterinary medicine, no guideline

for the treatment of pancreatic abscess has been established yet. In dogs, omentalization with abdominal closure is preferred over open peritoneal drainage even in sterile abscesses due to the possibility of complications in open peritoneal drainage such as septic peritonitis. This shows that treatment is often different between humans and pets, even if the disease may be the same. For pancreatic abscesses with positive bacteriological cultures in cats, surgical treatment is preferred over open peritoneal drainage. Nonetheless, regardless of the animal species, the primary aim of treatment of abscesses would be to control bacterial infection.

Several limitations in this study are worth noting. First, the origin of *S. aureus* has not been identified. Second, although no abnormalities were observed in the blood examination, radiographs, and ultrasonography, triaditis syndrome could not be excluded from the diagnosis of this cat because of the lack of histopathology.

This is the first report of feline pancreatic abscess due to *S. aureus* infection. FNA of the abscess and confirmation of the infection had allowed antibiotic treatment to start early. The early administration of antibiotic and abscess removal most probably contributed to the short hospitalization period. As the origin of the bacteria was unclear, more cases should be studied in detail in order to gather more relevant information related to pancreatic masses in veterinary medicine.

REFERENCES

1. Anderson, J. R., Cornell, K. K., Parnell, N. K. and Salisbury, S. K. 2008. Pancreatic abscess in 36 dogs: a retrospective analysis of prognostic indicators. *J. Am. Anim. Hosp. Assoc.* **44**: 171–179. [Medline] [CrossRef]
2. Bello, B. and Matthews, J. B. 2012. Minimally invasive treatment of pancreatic necrosis. *World J. Gastroenterol.* **18**: 6829–6835. [Medline] [CrossRef]
3. Bradley, E. L. 3rd. 1993. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Arch. Surg.* **128**: 586–590. [Medline] [CrossRef]
4. Branter, E. M. and Viviano, K. R. 2010. Multiple recurrent pancreatic cysts with associated pancreatic inflammation and atrophy in a cat. *J. Feline Med. Surg.* **12**: 822–827. [Medline] [CrossRef]
5. Cheng, A. G., DeDent, A. C., Schneewind, O. and Missiakas, D. 2011. A play in four acts: Staphylococcus aureus abscess formation. *Trends Microbiol.* **19**: 225–232. [Medline] [CrossRef]
6. Coleman, M. and Robson, M. 2005. Pancreatic masses following pancreatitis: pancreatic pseudocysts, necrosis, and abscess. *Compend. Contin. Educ. Pract. Vet.* **27**: 147–153.
7. Johnson, M. D. and Mann, F. A. 2006. Treatment for pancreatic abscesses via omentalization with abdominal closure versus open peritoneal drainage in dogs: 15 cases (1994–2004). *J. Am. Vet. Med. Assoc.* **228**: 397–402. [Medline] [CrossRef]
8. Kobayashi, S. D., Malachowa, N. and DeLeo, F. R. 2015. Pathogenesis of Staphylococcus aureus abscesses. *Am. J. Pathol.* **185**: 1518–1527. [Medline] [CrossRef]
9. Lee, M., Kang, J. H., Chang, D., Na, K. J. and Yang, M. P. 2015. Pancreatic abscess in a cat with diabetes mellitus. *J. Am. Anim. Hosp. Assoc.* **51**: 180–184. [Medline] [CrossRef]
10. Morris, D. O., Rook, K. A., Shofer, F. S. and Rankin, S. C. 2006. Screening of *Staphylococcus aureus, Staphylococcus intermedius*, and *Staphylococcus schleiferi* isolates obtained from small companion animals for antimicrobial resistance: a retrospective review of 749 isolates (2003−04). *Vet. Dermatol.* **17**: 332–337. [Medline] [CrossRef]
11. Son, T. T., Thompson, L., Serrano, S. and Seshadri, R. 2010. Surgical intervention in the management of severe acute pancreatitis in cats: 8 cases (2003−2007). *J. Vet. Emerg. Crit. Care (San Antonio)* **20**: 426–435. [Medline] [CrossRef]
12. VanEnkevort, B. A., O'Brien, R. T. and Young, K. M. 1999. Pancreatic pseudocysts in 4 dogs and 2 cats: ultrasonographic and clinicopathologic findings. *J. Vet. Intern. Med.* **13**: 309–313. [Medline] [CrossRef]
13. vanSonnenberg, E., Wittich, G. R., Goodacre, B. W., Casola, G. and D'Agostino, H. B. 2001. Percutaneous abscess drainage: update. *World J. Surg.* **25**: 362–369, discussion 370–372. [Medline] [CrossRef]
14. Widdison, A. L., Karanjia, N. D. and Reber, H. A. 1994. Antimicrobial treatment of pancreatic infection in cats. *Br. J. Surg.* **81**: 886–889. [Medline] [CrossRef]
15. Widdison, A. L., Karanjia, N. D. and Reber, H. A. 1994. Routes of spread of pathogens into the pancreas in a feline model of acute pancreatitis. *Gut* **35**: 1306–1310. [Medline] [CrossRef]
16. Widdison, A. L., Alvarez, C., Chang, Y. B., Karanjia, N. D. and Reber, H. A. 1994. Sources of pancreatic pathogens in acute pancreatitis in cats. *Pancreas* **9**: 536–541. [Medline] [CrossRef]
17. Xenoulis, P. G. and Steiner, J. M. 2012. Canine and feline pancreatic lipase immunoreactivity. *Vet. Clin. Pathol.* **41**: 312–324. [Medline] [CrossRef]