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Abdominal ultrasound has inconsistent agreement with subsequent surgery or necropsy findings in dogs and cats with septic peritonitis

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OBJECTIVE
To evaluate whether abdominal ultrasound correctly diagnosed septic peritonitis and correctly identified its causative lesion in dogs and cats.

ANIMALS
84 client-owned dogs and 10 cats that underwent an abdominal ultrasound and had confirmation of septic peritonitis via exploratory laparotomy or necropsy.

METHODS
This retrospective case series documented abdominal ultrasound findings, surgical or necropsy findings, and method for initial diagnosis of septic peritonitis, if different from surgery or necropsy. The surgical report and necropsy findings were compared to sonography results to confirm a diagnosis of septic peritonitis. The frequency at which sonography diagnosed septic peritonitis and its causative lesion was calculated for each type of lesion pathology and organ system. Secondary aims included evaluating the effect of patient characteristics (body weight and species) on sonographic results and whether lesion type or location affected mortality.

RESULTS
Most lesions causing septic peritonitis (70.2%) were gastrointestinal in origin and were nonneoplastic ulcerations or perforations (50%). Abdominal ultrasound diagnosed 56.3% of cases of subsequently confirmed septic peritonitis and correctly identified 67% of the causative lesions. Lesions of the gastrointestinal tract and ulcerations/perforations were the most frequent correct sonographic diagnoses and most likely to lead to a correct sonographic diagnosis of septic peritonitis. Lesions located in the hepatobiliary system and lesion types other than neoplasia or ulcerations/perforations were the most frequently missed by abdominal ultrasound.

CLINICAL RELEVANCE
Abdominal ultrasound often fails to diagnose septic peritonitis or the underlying causative lesion, and its accuracy depends on the affected organ and type of lesion.

Keywords: sepsis, septic peritonitis, abdominal ultrasound, ultrasound, exploratory laparotomy

Bacterial septic peritonitis is the most common cause of peritonitis in small animals and is associated with significant morbidity and mortality.[1–3] Sequelae of septic peritonitis include acute kidney injury or multiorgan dysfunction syndrome, and published mortality rates range from 30% to 60% in cats and 12.5% to 56% in dogs.[1,3–15] Secondary septic peritonitis, an intraperitoneal infection caused by an underlying disease process, is the most common etiology in small animal patients.[16] It is most frequently caused by bacterial leakage from gastrointestinal (GI) injury but can result from infection of any organ within the abdomen.[3,16] Regardless of the etiology, the general diagnostic workup of septic peritonitis often includes diagnostic imaging such as radiography, ultrasound, or CT. At our institution, ultrasound is the most utilized imaging modality in a clinical setting when abdominal pathology is suspected.

Septic peritonitis may be diagnosed by sonography; for example, if ultrasound identifies the presence of free gas or abdominal effusion that was not previously identified with subsequent confirmation of intracellular bacteria on cytology after abdominocentesis.

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Alternatively, a diagnosis of septic peritonitis may be made prior to a complete abdominal ultrasound if peritoneal effusion is initially detected and sampled by the primary clinician. This remains the simplest, most common, and most efficient method of diagnosis. In the latter scenario, ultrasound may still be performed for surgical planning and prognostication purposes. Downsides of abdominal ultrasound in these cases include a delay to surgical intervention, the costs associated with the procedure, a potential need for sedation in a compromised patient, and the necessity of a trained sonographer. More importantly, previous research has found conflicting evidence for the accuracy of ultrasound at diagnosing surgical lesions.[17–20] Those findings question the necessity of a complete abdominal ultrasound prior to surgical exploration of the abdomen, especially for cases in which a surgical disease process (eg, septic peritonitis) has already been diagnosed.

To our knowledge, although correlations between sonographic findings and surgical pathology have been previously investigated for several disease processes, there are no focused studies evaluating septic peritonitis, a condition in which timing to surgical intervention is of utmost importance.[18,20] Our primary aim was to compare abdominal ultrasound findings to pathology found at surgery or gross necropsy for dogs and cats with confirmed septic peritonitis. These results were compared to determine the frequency at which abdominal ultrasound correctly identified septic peritonitis prior to surgical or necropsy confirmation. We also sought to explore whether the lesion location or type affected sonographic diagnosis of septic peritonitis. Secondary goals included investigating whether patient species or weight affected sonographic results and whether there was an association between patient survival to discharge and lesion location or lesion type.

Methods

Data collection

This was a retrospective case series at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania. The electronic medical record system was searched from 2009 to 2023 to identify dogs and cats with septic peritonitis. Keywords searched included "septic peritonitis," "sepsis," and "septic abdomen." Animals were included if they had a full abdominal ultrasound followed by confirmation of septic peritonitis by either exploratory laparotomy or euthanasia/death with necropsy. Data collected from the medical record included species, age, sex and reproductive status, breed, body weight, abdominal ultrasound findings, surgical or necropsy results if applicable, final disposition (euthanized, died, or discharged alive), and results of abdominal effusion cytology and/or bacterial culture. Abdominal ultrasound was performed by board-certified radiologists (diplomates of the American or European College of Veterinary Radiology) or radiology residents using the Logiq S8, 9, and E10 (GE HealthCare) and Epiq 5 (Koninklijke Philips NV) ultrasound machines. Patients that underwent abdominal ultrasound during the working day (8:00 AM to 5:00 PM) were scanned by both a resident and diplomate, while afterhours ultrasound was performed by residents alone with subsequent diplomate evaluation of recorded cine-loops. All final ultrasound reports were cosigned by a diplomate. Animals were excluded if they had incomplete medical records, had no ultrasound prior to surgery, did not have surgical or gross necropsy confirmation of septic peritonitis, or developed septic peritonitis with prior known surgical intervention biasing the lesion localization.

Data interpretation

Septic peritonitis was diagnosed in the following ways: (1) by abdominal ultrasound if there was evidence of pneumoperitoneum or direct visualization of a lesion causing septic peritonitis (eg, visualized organ perforation), (2) at the time of surgery by direct visualization of intraperitoneal infection (eg, gastric contents or purulent exudate present in the peritoneum) or strong suspicion with confirmation by effusion cytology or a positive effusion bacterial culture, and (3) by gross necropsy if there was direct visualization of intraperitoneal infection (eg, gastric contents or purulent exudate present in the peritoneum) or strong suspicion of septic peritonitis with confirmation by histopathology or culture. Sonography was considered to have correctly diagnosed septic peritonitis when it met criteria for sonographic diagnosis of septic peritonitis and was later confirmed at the time of surgery or necropsy. Sonography was considered to have correctly identified the location of the causative lesion when the same organ was identified as the causative lesion of disease in the ultrasound report and either the surgical or necropsy report. The primary finding in the ultrasound report (ie, the sonographer's interpretation as the most likely cause of sepsis or the patient's clinical signs) was considered to agree with surgery or necropsy findings when the organ system and type of pathology identified by both modalities were the same. Correct sonographic diagnosis of septic peritonitis was not required to make a correct sonographic lesion identification.

Statistical analysis

Categorical variables are reported as counts and percentages. Most continuous variables were not normally distributed as determined visually and by the skewness and kurtosis tests; therefore, continuous variables are reported as median (range). The χ^2 or Fisher exact test was utilized to determine the relationship between 2 categorical variables depending on whether the frequency of observations per cell was ≤ 5. When a categorical variable (such as anatomic location of the lesion causing septic peritonitis) had > 2 categories, a Bonferroni correction was applied; Bonferroni-corrected *P* values are reported. Simple logistic regression was performed to determine whether binary outcomes such as survival or death were significantly associated with a continuous variable. A P value of < .05 was considered significant for all tests. All statistical evaluations were performed with a statistical software package (Stata version 14.0 for Mac; Stata Corp).

Results

Patient characteristics, methods of septic peritonitis diagnosis, and outcome

A total of 262 patients were identified during our initial search of the electronic medical record system. Reasons for exclusion included incomplete medical records, recent abdominal surgery, no abdominal ultrasound, or lack of definitive diagnosis of septic peritonitis (via surgery or necropsy). Ninety-four patients (10 cats and 84 dogs) were subsequently included in the study. The median age of all animals was 8 years (range, 6 months to 21 years). There were 47.9% (45 of 94) castrated males, 36.2% (34 of 94) spayed females, 9.6% (9 of 94) sexually intact males, and 6.4% (6 of 94) sexually intact females. Twenty-nine dog breeds were represented, with the most common breeds being mixed breed (34.5% [29 of 84]), Boxer (8.3% [7 of 84]), and Labrador Retriever (7.1% [6 of 84]). The most common cat species was domestic shorthair (80% [8 of 10]). The median dog weight was 26.3 kg (range, 1.8 to 69 kg), and the median cat weight was 4.1 kg (range, 2.7 to 5.2 kg). Seventy-four patients proceeded to surgery, while 20 were euthanized without surgery and underwent a diagnostic necropsy **(Figure 1)**. The overall mortality rate including euthanasia was 40.4% (38 of 94), and the mortality for those patients that underwent surgery was 24.3% (18 of 74).

Cytology was the most common method of initial identification of septic peritonitis in our patient population, utilized to make a diagnosis in 56.3% (53 of 94) of patients. Of these patients, abdominal ultrasound was also able to provide a sonographic diagnosis of septic peritonitis 50.9% (27 of 53) of the time. Other methods of initial diagnosis included abdominal ultrasound (29.8% [28 of 94]), exploratory laparotomy (11.7% [11 of 41]), and necropsy (3.2% [3 of 94]). Bacterial culture and sensitivity testing of abdominal effusion was performed in 61.7% (58 of 94) of patients, and 79.3% (46 of 58) were positive for either single or multiple bacterial organisms. *Escherichia coli* was the most detected bacterial isolate, identified in 54.3% (25 of 46) of patients, followed by *Enterococcus* spp (41.3% [19 of 46]) and *Staphylococcus* spp (19.6% [9 of 46]); 36.9% (17 of 46) of patients had > 1 bacterial species cultured.

Lesion location in dogs and cats with septic peritonitis

Lesions causing septic peritonitis were categorized on the basis of anatomic location and type of pathology. Locations of underlying causative lesions included the GI tract (stomach, small intestines, or large intestines), hepatobiliary system (liver or biliary tract), and other (kidneys, uterus, urinary bladder, or unidentified). Types of lesion pathology included nonneoplastic ulceration/perforation (eg, associated with a foreign body), neoplasia, non-GI abscess/infection, non-GI organ rupture (eg, gallbladder rupture), organ torsion, or unknown. Septic peritonitis was caused by a lesion of the GI tract in 70.2% (66 of 94) of patients, the hepatobiliary system in 16% (15 of 94) of patients, and other sites in 13.8% (13 of 94) of patients **(Table 1)**. The most common type of pathology leading to septic peritonitis was a nonneoplastic ulceration or perforation of the GI tract (50% [47 of 94]), followed by neoplasia (21.3% [20 of 94]), non-GI abscess or infection (11.7% [11 of 94]), non-GI organ rupture (6.4% [6 of 94]), organ torsion (2.1% [2 of 94]), and unknown (ie, primary septic peritonitis; 8.5% [8 of 94]; **Table 2**).

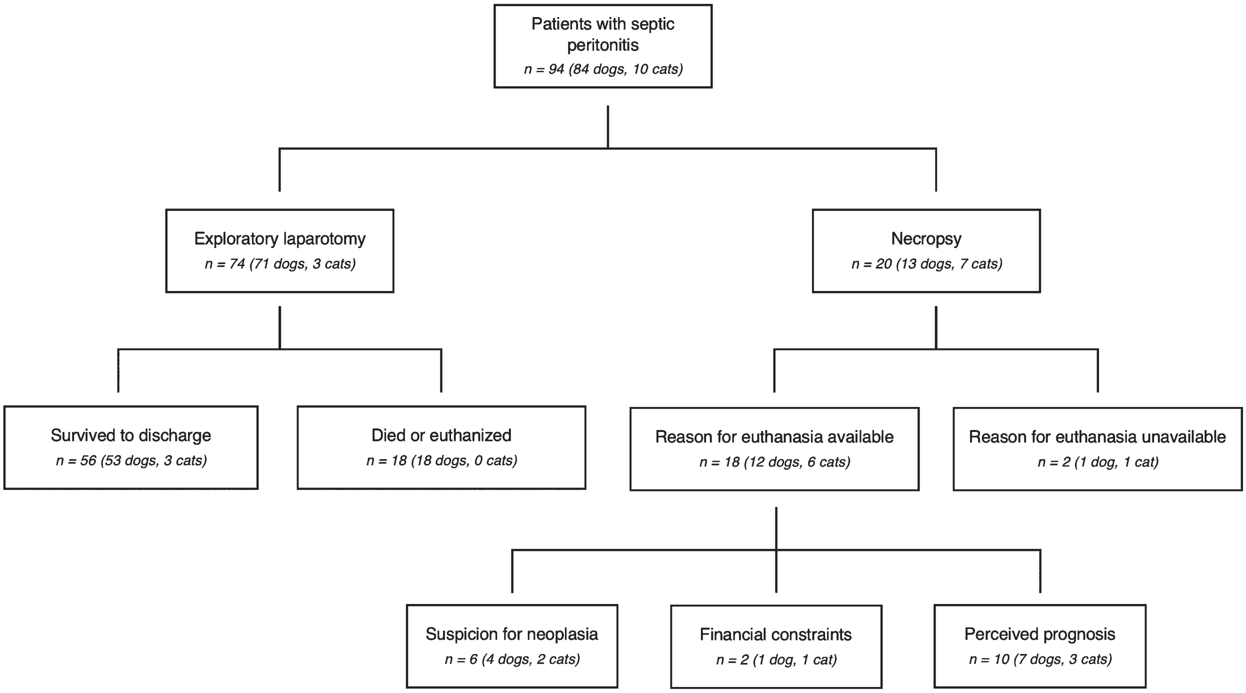

Figure 1—Outcome and reason for euthanasia for 94 dogs and cats with septic peritonitis.

Table 1—Lesion location, incidence, and species breakdown of 84 dogs and 10 cats with septic peritonitis.

Lesion location	No. (%) of incidences	Species
GI tract (n = 66)		
Small intestine	43 (45.7)	38 dogs, 5 cats
Stomach	14 (14.9)	13 dogs, 1 cat
Large intestine	9 (9.6)	8 dogs, 1 cat
Hepatobiliary (n = 15)		
Liver	9 (9.6)	8 dogs, 1 cat
Biliary tract	6 (6.4)	5 dogs, 1 cat
Other (n = 13)		
Peritoneum	4 (4.3)	4 dogs, 0 cats
Uterus	3 (3.2)	2 dogs, 1 cat
Kidney	2 (2.1)	2 dogs, 0 cats
Urinary bladder	2 (2.1)	2 dogs, 0 cats
Unidentified	2 (2.1)	2 dogs, 0 cats

GI = Gastrointestinal.

Table 2—Lesion type, incidence, and species breakdown of 84 dogs and 10 cats with septic peritonitis. Non-GI organs affected by abscess/infection or rupture included the kidney, gall bladder, liver, uterus, urinary bladder, and peritoneum.

Lesion type	No. (%) of incidences	Species
Perforation/ulceration (nonneoplastic)	47 (50.0)	41 dogs, 6 cats
Neoplasia	20 (21.3)	19 dogs, 1 cat
Abscess/infection (non-GI)	11 (11.7)	10 dogs, 1 cat
Organ rupture (non-GI)	6 (6.4)	5 dogs, 1 cat
Organ torsion	2 (2.1)	2 dogs, 0 cats
Unidentified	8 (8.5)	7 dogs, 1 cat

To allow for further analysis, lesion type (perforation/ulceration, neoplasia, and other) and lesion location (GI tract, hepatobiliary, and other organs) were grouped into 3 categories each.

Ability of abdominal ultrasound to correctly identify septic peritonitis or the underlying lesion

Abdominal ultrasound correctly identified septic peritonitis in 56.3% (53 of 94) of patients, and the underlying causative lesion was also identified in 79.3% (42 of 53) of these patients **(Figure 2)**. Of the patients that did not have sonographic diagnosis of septic peritonitis, 51% (21 of 41) still had the correct lesion location identified on ultrasound. The causative lesion was correctly identified by sonography in 67.0% (63 of 94) of all patients.

Abdominal ultrasound most frequently identified septic peritonitis when the causative lesion originated in the GI tract (68% correct), as compared to the hepatobiliary system (27% correct; *P* = .01) or other organs in the abdomen (31% correct; *P* = .04; **Figure 3**). In addition, abdominal ultrasound most frequently identified the correct anatomic location of the lesion (regardless of ability to detect septic peritonitis itself) for lesions of the GI tract (76% correct).

Abdominal ultrasound correctly identified septic peritonitis when the causative lesion was a nonneoplastic ulceration or perforation (70% correct) more frequently than all other nonneoplastic types of lesions (30% correct; *P* = .003; **Figure 4**). Similarly, abdominal ultrasound more frequently identified the correct anatomic location of the lesion causing septic peritonitis for nonneoplastic ulcerations/perforations (78.7% correct) than all other nonneoplastic causes (40.7% correct; *P* = .003). There was no difference in the ability of abdominal ultrasound to diagnose septic peritonitis (*P* = .76) or the underlying organ affected (*P* = .57) when comparing neoplastic lesions with nonneoplastic ulceration or perforation.

Other findings

There was no association between the type of lesion causing septic peritonitis (*P* = .71) or the location of the lesion (*P* = .21) and owners' decisions to euthanize prior to surgery. Similarly, there was no association

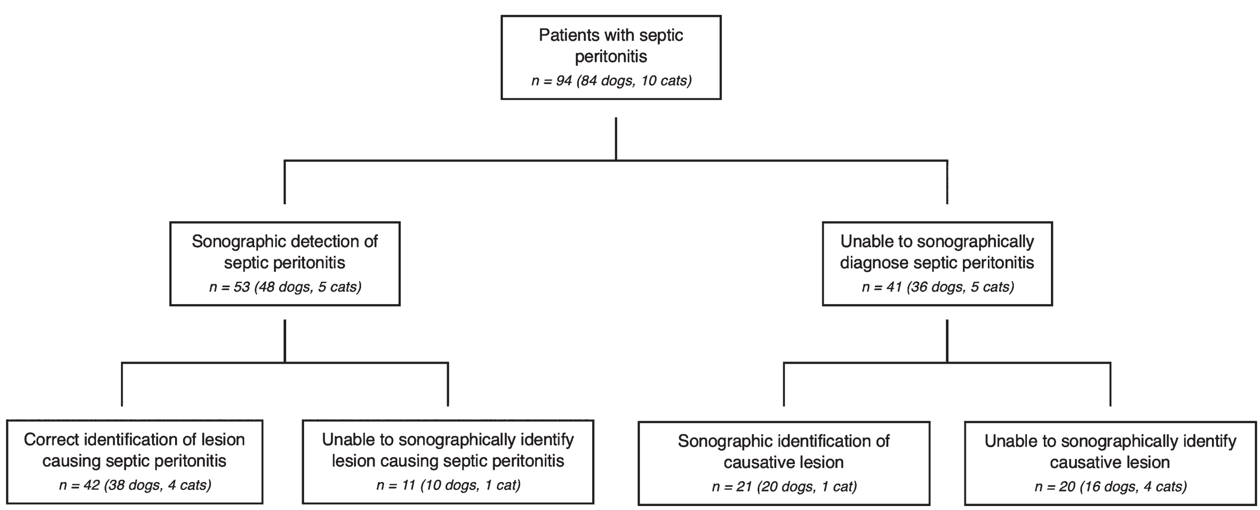

Figure 2—Ultrasonographic identification of septic peritonitis and the causative lesion in 94 dogs and cats with confirmed septic peritonitis at surgery or necropsy.

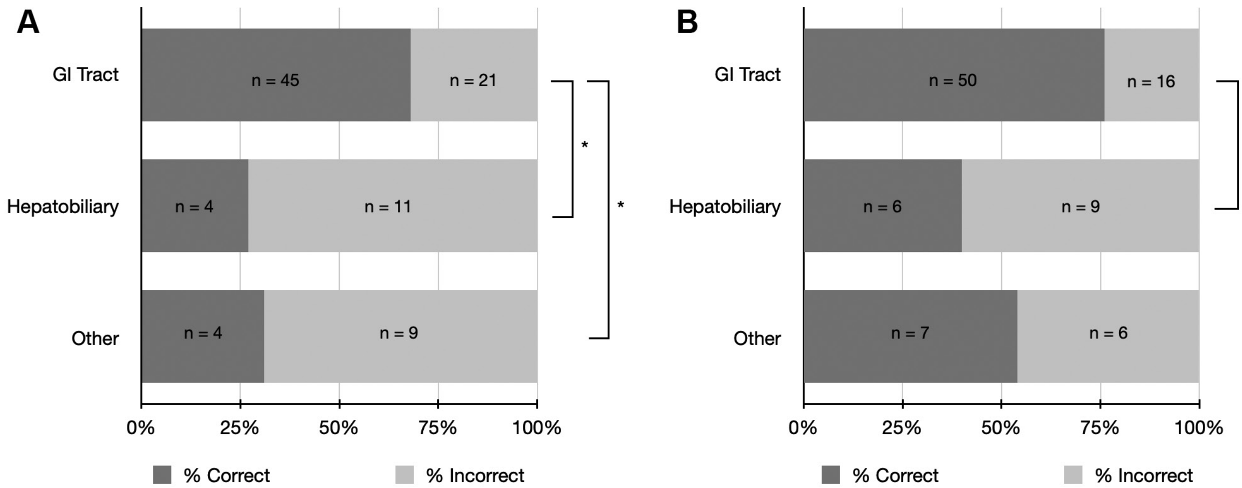

Figure 3—Ability of abdominal ultrasound to correctly diagnose septic peritonitis (panel A) and identify the underlying lesion (panel B) on the basis of confirmed anatomic lesion location at surgery/necropsy. n = Number of patients in each group out of a total of 94. Other = Lesions other than those of the gastrointestinal (GI) or hepatobiliary systems. Significance at *P* < .05 is indicated with an asterisk.

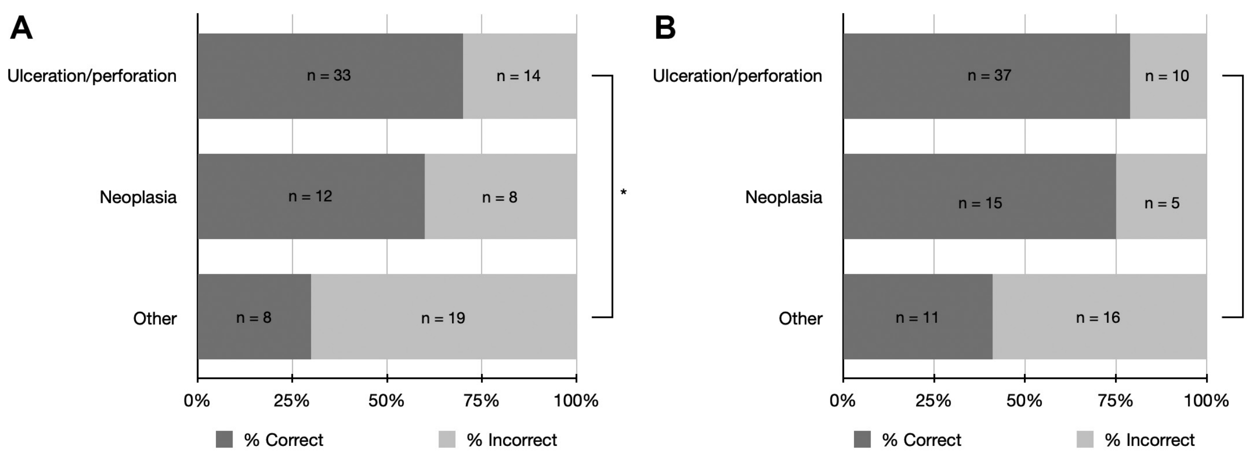

Figure 4—Ability of abdominal ultrasound to correctly diagnose septic peritonitis (panel A) and detect the underlying lesion by type of lesion (panel B) on the basis of confirmed lesion type at surgery/necropsy. n = Number of patients in each group out of a total of 94. Significance at *P* < .05 is indicated with an asterisk.

between the type of lesion and whether the patient survived to discharge (*P* = .6). However, patients were significantly more likely to survive to discharge when the location of the lesion causing septic peritonitis was the GI tract as compared to the hepatobiliary system (*P* = .021) or other organs (*P* = .027).

The weight of the patient did not affect the ability of abdominal ultrasound to identify either septic peritonitis (*P* = .248) or the correct location of the lesion causing septic peritonitis (*P* = .475). There was also no difference between dogs and cats in the ability of abdominal ultrasound to identify either septic peritonitis (*P* = .458) or the correct location of the lesion causing septic peritonitis (*P* = .194).

Discussion

This retrospective case series investigated the ability of abdominal ultrasound to diagnose septic peritonitis and detect the causative lesion in a population of dogs and cats that had subsequent surgical or necropsy confirmation of the diagnoses. These results corroborate previous studies that have found variable agreement between abdominal ultrasound results and surgical/histopathological findings, especially for lesions causing rupture of the GI tract. In a retrospective study of 100 dogs and cats, Pastore et al[18] found 64% agreement between ultrasound and intraoperative findings for all surgical abdominal diseases. A major discrepancy was observed between ultrasound and surgical findings in 25% of cases, and ulcerations/perforations/ruptures of the GI tract were the lesions most frequently missed. Another retrospective study[17] of spontaneous gastroduodenal rupture in dogs and cats found that abdominal ultrasound alone was only diagnostic for perforation in 9% of patients. A study[19] of small animals with confirmed GI perforation found that the sonographer who performed presurgical ultrasound listed "perforation" as a differential diagnosis in 74% of patients evaluated. Lastly, Abdellatif et al[20] compared ultrasound findings to subsequent cytological, histopathological, and surgical diagnoses in small animals presenting with acute abdomen syndrome. This group found that abdominal ultrasound was corroborated by cytology in 86.4% of lesions and by histopathology in 66.2% of lesions; surgical lesions were missed in 13.1% of patients. Given the sparse literature, differences in sonographic operator and technique, and advances in technology over the past two decades, it is challenging to draw definitive conclusions from these data. However, there is a trend toward significant inconsistency between abdominal sonography and eventual findings at the time of surgery or histopathology.

Our finding that ulcerations/perforations and GI tract lesions were the most correctly identified is likely because nontraumatic transmural injury to the GI tract often leads to pneumoperitoneum, which by itself is strong evidence of viscous rupture and leakage of GI contents, even in the absence of a definitive location of injury. Septic peritonitis from injury to other organs does not frequently lead to accumulation of free gas and is therefore more challenging to diagnose. The presence of peritonitis or echogenic effusion is suggestive of intra-abdominal inflammation but not diagnostic for septic peritonitis.[21] Spontaneous pneumoperitoneum secondary to other causes including splenic necrosis, urinary bladder rupture, or idiopathic causes has also been reported.[22–24] Although definitive numbers are not available for small animal sonography, a human meta-analysis[25] found that ultrasound was 91% sensitive and 96% specific for peritoneal free gas. Experimentally induced pneumoperitoneum in healthy Beagles can be detected reliably with volumes of gas as low as 0.2 to 0.4 mL.[26,27] However, free gas can still be missed if the volume is too small to detect, if the lesion has been plugged by omentum or other viscera, or due to operator skill. Additionally, not all GI perforations lead to pneumoperitoneum.[21]

Our data did not find that patient species or body weight affected ultrasound results. Our results support earlier work that found no effect of patient size on the sensitivity of ultrasound to detect surgical abdominal lesions or the location of solitary hepatic tumors.[18,28] In human sonography, patient weight can have a detrimental effect on image quality in several ways. Fat tissue can cause attenuation of ultrasound, lowering the image quality of deeper structures.[29] Obesity leads to an increased distance from the probe to the organs being evaluated and necessitates the use of lower frequency probes, which have a decreased resolution.[29] Given that we did not collect body condition data for our patients, it is unknown whether patient size suggested overconditioning or rather simply reflected normal canine variations in size. Nevertheless, it is still reasonable to assume that larger dogs would be more difficult to image given the distance that the ultrasound beam needs to travel. Since most patients in our study had GI tract lesions and this type of lesion most often led to a correct diagnosis of septic peritonitis, it is possible that the location of the GI tract being generally more superficial than other organ systems contributed to this finding. Alternatively, pneumoperitoneum may be easy to detect regardless of size or body condition since gas rises and will usually settle closer to the ultrasound probe.

Septic peritonitis can be a devastating diagnosis for both the patient and pet owner, as it is associated with significant morbidity and mortality. We reported an overall mortality rate of 40.4%, including those with an initial decision for euthanasia/necropsy over surgery, and a 24.3% mortality rate of those patients that proceeded to exploratory laparotomy. These numbers are similar to those that have been previously reported.[1,3–15] The finding that animals with GI lesions were more likely to survive to discharge may be because these types of lesions often cause more easily recognizable clinical signs, can be identified earlier in the disease process, and are often amenable to definitive surgical intervention.

When considering an abdominal ultrasound in a clinical context, many factors must be evaluated by the attending clinician when deciding on the need for further imaging, especially if a diagnosis of septic peritonitis has already been achieved. We found that abdominal ultrasound was unable to verify a diagnosis of septic peritonitis in almost half of the patients that had initial diagnosis of septic peritonitis via cytology of abdominal effusion. Acquiring a full ultrasound study may add a significant time delay to the onset of surgery, and human research has shown that delays in achieving surgical source control for intra-abdominal and GI sources of sepsis are associated with increased risk of death.[30–33] Additionally, given the degree of pain associated with peritonitis, ultrasound often requires heavy sedation, which adds additional risks for a potentially hemodynamically compromised patient. Finally, imaging can add further financial burden in a surgical disease already associated with some of the highest costs in the industry due to the intensive nature of these cases, high morbidity, and frequently extensive hospitalization requirements. Cytology of septic peritoneal effusion remains the simplest and most accurate method of diagnosing septic peritonitis, and it is not unexpected that abdominal ultrasound had poor sensitivity for septic peritonitis in our study. In contrast to these downsides, abdominal ultrasound can provide benefits to both the surgeon and pet owner. Given the wide variety of diseases that can lead to septic peritonitis, ultrasound can help with surgical planning and provide prognostic information, including the identification of secondary diseases such as neoplasia. Diagnosing a GI source of sepsis (eg, by diagnosing pneumoperitoneum), which was associated with survival in our study, is also a benefit of sonography. Yet free gas is something that may be able to be detected by less invasive or expensive tests such as point-of-care ultrasound or radiography. Our data regarding the performance of abdominal ultrasound in patients with septic peritonitis contribute to this decision-making process and may suggest that abdominal ultrasound is a less necessary and useful step if not needed for the initial diagnosis.

There were several limitations of this study. Our small study population, particularly for cats, may have prevented detection of differences between groups due to a lack of statistical power. The fact that septic peritonitis can cause many concurrent sonographic lesions may limit the applicability of our results to other intra-abdominal diseases with less complex pathology. Due to the retrospective nature of the study, case management including the decision to pursue imaging was not standardized. Our results may differ from previous studies due to patient differences, advances in equipment, or the technique of the operator. Within our patient population, ultrasound examinations were performed with nonstandardized methodology, and differences in operator experience, skill, or equipment could limit the generalizability of our results. Despite these limitations, the heterogeneity in sonographic technique does reflect the real-world differences in clinical approach that can be found throughout different types of veterinary practices. A prospective study evaluating the costs and benefits of abdominal ultrasound performed after diagnosis of septic peritonitis would allow more definitive conclusions about its usefulness.

In conclusion, abdominal ultrasound was able to correctly diagnose septic peritonitis in just over half of all study patients but was better at identifying the organ affected by the causative lesion. The greatest utility of ultrasound is for septic peritonitis originating from the GI tract, especially if the lesion is a nonneoplastic ulceration or perforation. The ability of ultrasound to diagnose septic peritonitis resulting from non-GI lesions was poor, possibly due to the nonspecific and heterogenous nature of sonographic findings with these disease processes, as compared to GI lesions that often result in pneumoperitoneum. Lesions aside from neoplasia or nonneoplastic ulceration/perforation were similarly difficult to diagnose. These results emphasize the need for careful patient selection when imaging patients already diagnosed with septic peritonitis and suggest that a uniform requirement for abdominal ultrasound may not provide much additional benefit.

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References

1. Anderson T, Beever L, Hall J, et al. Outcome following surgery to treat septic peritonitis in 95 cats in the United Kingdom. *J Small Anim Pract*. 2021;62(9):744–749. doi:10.1111/jsap.13346
2. Bentley A, Balakrishnan A. Peritonitis. In: LR Aronson, ed. *Small Animal Surgical Emergencies*. 2nd ed. Wiley Blackwell; 2022:119–140.
3. Shipov A, Lenchner I, Milgram J, et al. Aetiology, clinical parameters and outcome in 113 dogs surgically treated for septic peritonitis (2004–2020). *Vet Rec*. 2023;192(6):e2134. doi:10.1002/vetr.2134
4. Snipes MM, Schmiedt CW, Lourenço BN, et al. Acute kidney injury is common in dogs with septic peritonitis and is associated with increased mortality. *J Am Vet Med Assoc*. Published online August 15, 2023. doi:10.2460/javma.23.04.0184
5. Kenney EM, Rozanski EA, Rush JE, et al. Association between outcome and organ system dysfunction in dogs with sepsis: 114 cases (2003–2007). *J Am Vet Med Assoc*. 2010;236(1):83–87. doi:10.2460/javma.236.1.83
6. Ruthrauff CM, Smith J, Glerum L. Primary bacterial septic peritonitis in cats: 13 cases. *J Am Anim Hosp Assoc*. 2009;45(6):268–276. doi:10.5326/0450268
7. Parsons KJ, Owen LJ, Lee K, Tivers MS, Gregory SP. A retrospective study of surgically treated cases of septic peritonitis in the cat (2000–2007). *J Small Anim Pract*. 2009;50(10):518–524. doi:10.1111/j.1748-5827.2009.00790.x
8. Costello MF, Drobatz KJ, Aronson LR, King LG. Underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis: 51 cases (1990–2001). *J Am Vet Med Assoc*. 2004;225(6):897–902. doi:10.2460/javma.2004.225.897
9. Mueller MG, Ludwig LL, Barton LJ. Use of closed-suction drains to treat generalized peritonitis in dogs and cats: 40 cases (1997–1999). *J Am Vet Med Assoc*. 2001;219(6):789–794. doi:10.2460/javma.2001.219.789
10. Barfield DM, Tivers MS, Holahan M, Welch K, House A, Adamantos SE. Retrospective evaluation of recurrent secondary septic peritonitis in dogs (2000–2011): 41 cases. *J Vet Emerg Crit Care (San Antonio)*. 2016;26(2):281–287. doi:10.1111/vec.12413
11. Marshall H, Sinnott-Stutzman V, Ewing P, Bracker K, Kalis R, Khorzad R. Effect of peritoneal lavage on bacterial isolates in 40 dogs with confirmed septic peritonitis. *J Vet Emerg Crit Care (San Antonio)*. 2019;29(6):635–642. doi:10.1111/vec.12893
12. Troìa R, Mascalzoni G, Calipa S, Magagnoli I, Dondi F, Giunti M. Multiorgan dysfunction syndrome in feline sepsis: prevalence and prognostic implication. *J Feline Med Surg*. 2019;21(6):559–565. doi:10.1177/1098612X18792106
13. Mouat EE, Davis GJ, Drobatz KJ, Wallace KA. Evaluation of data from 35 dogs pertaining to dehiscence following intestinal resection and anastomosis. *J Am Anim Hosp Assoc*. 2014;50(4):254–263. doi:10.5326/JAAHA-MS-6111
14. Cortellini S, Seth M, Kellett-Gregory LM. Plasma lactate concentrations in septic peritonitis: a retrospective study of 83 dogs (2007–2012). *J Vet Emerg Crit Care (San Antonio)*. 2015;25(3):388–395. doi:10.1111/vec.12234
15. Dickinson AE, Summers JF, Wignal J, Boag AK, Keir I. Impact of appropriate empirical antimicrobial therapy on outcome of dogs with septic peritonitis. *J Vet Emerg Crit Care (San Antonio)*. 2015;25(1):152–159. doi:10.1111/vec.12273
16. Scotti KM, Koenigshof A, Sri-Jayantha LSH, et al. Prognostic indicators in cats with septic peritonitis (2002–2015): 83 cases. *J Vet Emerg Crit Care (San Antonio)*. 2019;29(6):647–652. doi:10.1111/vec.12896
17. Hinton LE, McLoughlin MA, Johnson SE, Weisbrode SE. Spontaneous gastroduodenal perforation in 16 dogs and seven cats (1982–1999). *J Am Anim Hosp Assoc*. 2002;38(2):176–187. doi:10.5326/0380176
18. Pastore GE, Lamb CR, Lipscomb V. Comparison of the

results of abdominal ultrasonography and exploratory laparotomy in the dog and cat. *J Am Anim Hosp Assoc*. 2007;43(5):264–269. doi:10.5326/0430264
19. Boysen SR, Tidwell AS, Penninck DG. Ultrasonographic findings in dogs and cats with gastrointestinal perforation. *Vet Radiol Ultrasound*. 2003;44(5):556–564. doi:10.1111/j.1740-8261.2003.tb00507.x
20. Abdellatif A, Kramer M, Failing K, von Pückler K. Correlation between preoperative ultrasonographic findings and clinical, intraoperative, cytopathological, and histopathological diagnosis of acute abdomen syndrome in 50 dogs and cats. *Vet Sci*. 2017;4(3):39. doi:10.3390/vetsci4030039
21. Stieger-Vanegas SM, Frank PM. Peritoneal space. In: Thrall DE, ed. *Textbook of Veterinary Diagnostic Radiology*. 7th ed. Elsevier; 2018:764–791. doi:10.1016/B978-0-323-48247-9.00051-6
22. Mehl ML, Seguin B, Norrdin RW, Geddes S, Withrow SJ. Idiopathic pneumoperitoneum in a dog. *J Am Anim Hosp Assoc*. 2001;37(6):549–551. doi:10.5326/15473317-37-6-549
23. Saunders WB, Tobias KM. Pneumoperitoneum in dogs and cats: 39 cases (1983–2002). *J Am Vet Med Assoc*. 2003;223(4):462–468. doi:10.2460/javma.2003.223.462
24. Rubanick JV, Breiteneicher AH, Thieman-Mankin K. Large-volume pneumoperitoneum and septic peritonitis secondary to splenic abscess rupture in a dog. *Can Vet J*. 2020;61(2):138–141.
25. Jiang L, Wu J, Feng X. The value of ultrasound in diagnosis of pneumoperitoneum in emergent or critical conditions: a meta-analysis. *Hong Kong J Emerg Med*. 2019;26(2):111–117. doi:10.1177/1024907918805668
26. Choi H, Lee Y, Park K, Yeon S, Lee H. Sonographic detection of small amounts of free peritoneal gas in Beagle dogs. *J Vet Med Sci*. 2012;74(4):491–494. doi:10.1292/jvms.11-0375
27. Kim SY, Park KT, Yeon SC, Lee HC. Accuracy of sonographic diagnosis of pneumoperitoneum using the enhanced peritoneal stripe sign in Beagle dogs. *J Vet Sci*. 2014;15(2):195–198. doi:10.4142/jvs.2014.15.2.195
28. Wormser C, Reetz JA, Giuffrida MA. Diagnostic accuracy of ultrasound to predict the location of solitary hepatic masses in dogs. *Vet Surg*. 2016;45(2):208–213. doi:10.1111/vsu.12436
29. Machann J, Horstmann A, Born M, Hesse S, Hirsch FW. Diagnostic imaging in obesity. *Best Pract Res Clin Endocrinol Metab*. 2013;27(2):261–277. doi:10.1016/j.beem.2013.02.003
30. Azuhata T, Kinoshita K, Kawano D, et al. Time from admission to initiation of surgery for source control is a critical determinant of survival in patients with gastrointestinal perforation with associated septic shock. *Crit Care*. 2014;18(3):R87. doi:10.1186/cc13854
31. Buck DL, Vester-Andersen M, Møller MH; Danish Clinical Register of Emergency Surgery. Surgical delay is a critical determinant of survival in perforated peptic ulcer. *Br J Surg*. 2013;100(8):1045–1049. doi:10.1002/bjs.9175
32. Reitz KM, Kennedy J, Li SR, et al. Association between time to source control in sepsis and 90-day mortality. *JAMA Surg*. 2022;157(9):817–826. doi:10.1001/jamasurg.2022.2761
33. Song SR, Liu YY, Guan YT, et al. Timing of surgical operation for patients with intra-abdominal infection: a systematic review and meta-analysis. *World J Gastrointest Surg*. 2023;15(10):2320–2330. doi:10.4240/wjgs.v15.i10.2320