Article

Risk factors of enterococcal bacteriuria in cats: A retrospective study

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Abstract

Objective

To determine if factors associated with urothelial damage and inflammation, including urinary catheterization, urinary obstruction, and urolithiasis are associated with the presence of enterococcal bacteriuria in cats.

Animals

Thirty-one cats with *Enterococcus* spp. bacteriuria and 31 cats with *Escherichia coli* bacteriuria.

Procedure

A retrospective case-control study with cases and controls identified by records search for *Enterococcus* spp. (case) and *E. coli* (control) bacteriuria from August 1, 2014 to July 31, 2019. Cases and controls were balanced with respect to average age. Binary logistic regression was used to estimate and test whether the odds of having *Enterococcus* spp. bacteriuria (instead of *E. coli*) were associated with the presence of any characteristic.

Results

Urinary catheterization, urinary obstruction, and urolithiasis were not observed more often in *Enterococcus* cases *versus E. coli* controls (19% *versus* 25%, $P = 0.543$; 19% *versus* 32%, $P = 0.244$; and 16% *versus* 16%, $P = 1$, respectively). Signs of lower urinary tract disease were significantly less common in *Enterococcus* cases than in *E. coli* controls (OR: 0.30; 95% CI: 0.10 to 0.83, $P = 0.02$). Hematuria was significantly less common in cases than controls ($P = 0.048$).

Conclusion

No association was identified between urinary catheterization, urolithiasis, or any other comorbidities (hyperthyroidism, chronic kidney disease) and enterococcal bacteriuria in cats.

Clinical relevance

Unlike in humans and dogs, urothelial damage and inflammation caused by factors such as urinary catheterization and urolithiasis may not be the mechanism for enterococcal bacteriuria in cats.

Résumé

Facteurs de risque de bactériurie à entérocoque chez le chat : une étude rétrospective

Objectif

Déterminer si les facteurs associés aux lésions et à l'inflammation urothéliales, y compris le cathétérisme urinaire, l'obstruction urinaire et les lithiases urinaires, sont associés à la présence de bactériurie à entérocoque chez le chat.

Animaux

Trente et un chats avec bactériurie à *Enterococcus* spp. et 31 chats atteints de bactériurie à *Escherichia coli*.

Procédure

Une étude cas-témoins rétrospective avec des cas et des témoins identifiés par la recherche de dossiers pour bactériurie à *Enterococcus* spp. (cas) et à *E. coli* (témoin) du 1er août 2014 au 31 juillet 2019. Les cas et les témoins étaient équilibrés par rapport à l'âge moyen. La régression logistique binaire a été utilisée pour estimer et tester si la probabilité d'avoir une bactériurie à *Enterococcus* spp. (au lieu d'*E. coli*) était associée à la présence de n'importe quelle caractéristique.

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Résultats

Le cathétérisme urinaire, l'obstruction urinaire et la lithiase urinaire n'ont pas été observés plus souvent chez les cas avec *Enterococcus* spp. par rapport aux témoins avec *E. coli* (19 % vs 25 %, *P* = 0,543; 19 % vs 32 %, *P* = 0,244; et 16 % vs 16 %, *P* = 1, respectivement). Les signes de maladie des voies urinaires inférieures étaient significativement moins fréquents chez les cas à *Enterococcus* que chez les témoins à *E. coli* (OR : 0,30; IC à 95 % : 0,10 à 0,83, *P* = 0,02). L'hématurie était significativement moins fréquente chez les cas que chez les témoins (*P* = 0,048).

Conclusion

Aucune association n'a été identifiée entre le cathétérisme urinaire, la lithiase urinaire ou toute autre comorbidité (hyperthyroïdie, maladie rénale chronique) et la bactériurie à entérocoque chez le chat.

Pertinence clinique

Contrairement aux humains et aux chiens, les lésions urothéliales et l'inflammation causées par des facteurs tels que le cathétérisme urinaire et la lithiase urinaire peuvent ne pas être le mécanisme pour la bactériurie à entérocoque chez les chats.

(Traduit par Dr Serge Messier)

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Introduction

Enterococci are Gram-positive bacteria that possess both natural and acquired resistance mechanisms predisposing humans and animals to multi-drug resistant infections that can be challenging to manage (1,2). In cats, the bacteria colonize the urinary tract after fecal contamination of the perineal region and subsequent bacterial ascension through the urethra. Enterococci cause approximately 5% of uncomplicated urinary tract infections (UTI) and 11% of complicated UTI in humans (3) and approximately 10% of uncomplicated UTI and up to 25% of recurrent UTI in dogs (4–6). In contrast, the prevalence of *Enterococcus* is higher in cats, with *Enterococcus* causing up to 43% of occult feline bacteriuria cases and up to 27% of UTI in symptomatic patients (7–9). The reason for the higher prevalence in cats is currently not known. Identifying variables that predispose cats to *Enterococcus* bacteriuria may allow for these variables to be corrected or avoided thereby preventing *Enterococcus* bacteriuria rather than relying on antibiotics to treat multidrug resistant infections.

In humans, enterococcal bacteriuria commonly occurs secondary to urinary catheter induced inflammation and subsequent fibrinogenuria because increased urine fibrinogen is both a source of nutrition and a binding agent for *Enterococcus* during biofilm formation (10,11). In dogs, a recent retrospective study identified lower urinary tract neoplasia, lower urinary tract anatomic abnormalities, and urolithiasis as enterococcal bacteriuria risk factors (12). A subsequent study associated these risk factors with fibrinogenuria in the dog providing circumstantial evidence that, as in humans, fibrinogenuria is permissive for *Enterococcus* bacteriuria in dogs (13).

The primary aim of this study was to assess whether clinicopathologic factors causing urothelial damage and inflammation, including urinary catheterization, urinary tract obstruction, and urolithiasis were associated with the presence of enterococcal bacteriuria in cats by comparing these variables between cats diagnosed with *Escherichia coli (E. coli)* and *Enterococcus* bacteriuria. We hypothesized that these factors would be associated with an increased risk of enterococcal bacteriuria in cats based on the previously stated findings in both humans and dogs. A sub-aim was to explore whether other comorbidities previously associated with enterococcal bacteriuria in cats in a non-controlled retrospective study (14) could be verified as risk factors in a case-control study. These variables included chronic kidney disease, hyperthyroidism, presence of a cystostomy tube, and spinal dysfunction.

Materials and methods

Study design and population

This was a single center retrospective case-control study with cases and controls selected so the 2 groups would be balanced with respect to age. The cases were not directly matched to controls so that comparisons could be made when data gaps existed in a medical record. The University of Wisconsin Veterinary Care Microbiology Service urine culture records from August 1, 2014 to July 31, 2019 were reviewed to identify cats with significant *Enterococcus* bacterial growth on urine culture (case subjects) and cats with significant *E. coli* growth (control subjects). *Escherichia coli* was used as the control organism because it is the most common cause of significant bacteriuria in cats (9,15,16). Significant bacterial growth was defined as individual bacterial species colony-forming units (cfu) $>$ 100 000 cfu/mL for all urine sample collection methods. For cats with growth of multiple bacterial species, significant growth was assessed using counts for the individual species of bacteria. Cats with more than one positive urine culture during the study timeline were only included once, with the most recent episode of bacteriuria recorded along with all past medical history.

Study variables

Demographic data collected and recorded for each subject included age, sex, and weight. If available, historical clinical data spanning each cat's entire medical record. This included (presence/absence of stranguria, dysuria, pollakiuria, and gross hematuria, or evidence of recurrent bacteriuria defined as $\geq$ 2 episodes in the last 6 mo or $\geq$ 3 episodes in the last year) and comorbidity data [presence/absence of endocrine disease (diabetes mellitus or hyperthyroidism), corticosteroid administration, urinary obstruction or a history of feline idiopathic

Table 1. Demographic and clinicopathologic characteristics for *Enterococcus* spp. cases and *Escherichia coli* controls.

Characteristic	*P*-value	Case	Control	OR (95% CI)
Sex				
Neutered male	0.091	6 (*n* = 19)	12 (*n* = 39)	— ref —
Spayed female		25 (*n* = 81)	19 (*n* = 61)	2.63 (0.86, 8.77)
Weight[a]				
≤ 5 kg	0.226	24 (*n* = 77)	19 (*n* = 63)	— ref —
> 5 kg		7 (*n* = 23)	11 (*n* = 37)	0.50 (0.16, 1.53)
Resistance pattern				
Susceptible	0.116	9 (*n* = 29)	15 (*n* = 48)	— ref —
Resistant		22 (*n* = 71)	16 (*n* = 52)	2.29 (0.82, 6.73)
Co-infection				
0 (none)	0.348	23 (*n* = 74)	26 (*n* = 84)	— ref —
1 additional		8 (*n* = 26)	5 (*n* = 16)	1.81 (0.53, 6.73)
Clinical signs of LUTD[b]				
No	0.020	22 (*n* = 71)	13 (*n* = 42)	— ref —
Yes		9 (*n* = 29)	18 (*n* = 58)	0.30 (0.10, 0.83)

OR — Odds ratio; CI — Confidence interval.
[a] One missing control (weight unknown).
[b] Lower urinary tract disease.

cystitis, urinary catheterization, renal azotemia, urolithiasis, or urinary incontinence] were collected and recorded. Recent use of antibiotics within 30 d of the bacteriuria episode was also recorded. Lastly, clinicopathologic data [urine specific gravity, presence/absence of proteinuria or hematuria (urine dipstick), presence/absence of ≥ 1 to 3 white blood cells or epithelial cells per high power field (hpf) (urine sediment)] and microbiologic data (number of bacterial isolates or number of antibiotic classes to which the isolates had resistance) were collected and recorded. The presence/absence of red blood cells (urine sediment) was also recorded but not used in the analysis because the urine sediment was not consistently recorded in the medical record and the available results aligned with urine dipstick analysis. Antibiotic classes included penicillin, cephalosporin, fluoroquinolone, aminoglycoside, carbapenum, and other. Cats with bacteriuria and clinical signs of lower urinary tract disease (pollakiuria, dysuria, stranguria, gross hematuria) were classified as having urinary tract infections, whereas all others were classified as having subclinical bacteriuria. Isolates were identified using biochemical identification methods and/or a commercial identification system (Vitek 2; BioMérieux, Durham, North Carolina, USA). A commercial system was also used to determine each isolate's antibiotic minimal inhibitory concentrations (Sensititre; TREK Diagnostic Systems, Cleveland, Ohio, USA) and interpreted based on the Clinical and Laboratory Standards Institute guidelines for antimicrobial susceptibility (17–19).

Statistical analyses

Frequencies and percentages were used to describe demographic characteristics and risk factors of interest for cases and controls. Binary logistic regression was used to test whether the odds of having *Enterococcus* bacteriuria were associated with the presence of any given characteristic. Significance was assessed using a likelihood ratio test (LRT) from the logistic regression model, and odds ratios (OR) determined by inverting the test. Parzen's method was used when a particular characteristic or risk factor did not occur or did occur for 100% of a particular group (20). Analyses were performed using *R* (v.4.0.2) (R Core Team. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria, 2020). To assess the study's primary aim that the 3 clinicopathologic factors: urinary catheterization, urolithiasis, and urinary obstruction, were associated with the presence of enterococcal bacteriuria statistical significance was set at the 0.05 level (2-sided). Other secondary aim comparisons were exploratory and hence the significance was not adjusted for multiplicity.

Results

In total, 90 cases of *Enterococcus* bacteriuria were identified from 43 unique cats within the study period. Of the 43 unique cats, 12 were removed from the study as they did not have measurable growth of *Enterococcus* bacteriuria on urine culture, leaving 31 unique cats. Of these 31 bacteriuria cases, 26 were identified as *Enterococcus faecalis (E. faecalis),* 3 were *Enterococcus faecium (E. faecium),* and 2 were unspecified *Enterococcus* species. Thirty-one *E. coli* control bacteriuria cases were selected from 194 cases of significant *E. coli* bacteriuria identified within the same study period. Cases and controls groups were balanced with respect to average age. The average age was 11.2 ± 5.44 y for cases and 11.2 ± 5.24 y for controls with a standardized mean difference of 1%. Although cases and controls were not selected to be balanced with respect to urine collection method, cystocentesis was the collection method for 28 case and 26 control samples, and transurethral catheterization was the collection method for 3 case and 5 control samples. All cases and controls had > 100 000 cfu/mL of bacteria detected by either of these urine collection methods.

Demographically, there was not a significant difference ($P = 0.091$) in the distribution of spayed female and neutered male cats between the case and control groups. In addition, there was not a significant difference ($P = 0.226$) in weight classes (≤ 5 kg and > 5 kg) between cases and controls. Clinically, signs of lower urinary tract disease were significantly less common in *Enterococcus* cases than in *E. coli* controls (29% *versus*

Table 2. Comparison of bacteriuria risk factors for *Enterococcus* spp. cases and *Escherichia coli* controls.

Characteristic	*P*-value	Case	Control	OR (95% CI)
History of catheterization				
No	0.543	25 (*n* = 81)	23 (*n* = 74)	— ref —
Yes		6 (*n* = 19)	8 (*n* = 26)	0.69 (0.20, 2.28)
Urinary obstruction/feline idiopathic cystitis				
No	0.244	25 (*n* = 81)	21 (*n* = 68)	— ref —
Yes		6 (*n* = 19)	10 (*n* = 32)	0.50 (0.15, 1.59)
Uroliths				
No	1	26 (*n* = 84)	26 (*n* = 84)	— ref —
Yes		5 (*n* = 16)	5 (*n* = 16)	1.00 (0.25, 4.00)

OR — Odds ratio; CI — Confidence interval.

58%; OR: 0.30; 95% CI: 0.10 to 0.83, $P = 0.020$). There was not a significant difference in the detection of bacterial co-infection nor resistance to ≥ 1 antibiotic class between cases and controls (26% *versus* 16%, $P = 0.348$ and 71% *versus* 52%, $P = 0.116$, respectively) (Table 1).

The presence of comorbidities such as a history of urinary catheterization, urinary obstruction, and urolithiasis were not significantly different between cases and controls (19% *versus* 25%, $P = 0.543$; 19% *versus* 32%, $P = 0.244$; and 16% *versus* 16%, $P = 1$, respectively) (Table 2). In addition, there was not a significant difference in the number of cats with hyperthyroidism or chronic kidney disease between case and control groups (25% *versus* 13%, $P = 0.195$; and 52% *versus* 48%, $P = 0.800$, respectively). No cats from either group had a cystostomy tube or spinal dysfunction. Clinicopathologically, hematuria was significantly less common in *Enterococcus* cases than *E. coli* controls (65% *versus* 90%, $P = 0.048$), although this association was based on a limited subset of the original sample (23 cases and 20 controls). Additional comparisons determined that 89% of *Enterococcus* cases and 67% of *E. coli* controls had bacteria detected on urine sediment, and urine pH was similar between the 2 groups (pH < 7 in 81% of cases and 75% of controls).

Discussion

This study determined that factors causing urothelial damage and inflammation, such as urinary catheterization, urinary obstruction, and urolithiasis, are not associated with an increased risk of enterococcal bacteriuria in cats. Given the importance of inflammation and subsequent fibrinogenuria for the development of *Enterococcus* bacteriuria in humans and dogs (12,13), possible explanations for the difference in cats include: i) the risk factors examined in this study do not cause fibrinogenuria in cats; ii) fibrinogenuria from inflammation is the mechanism for enterococcal bacteriuria in cats, but the small sample size and/or retrospective design of this study did not allow us to identify an association; or iii) fibrinogenuria is not the mechanism promoting enterococcal bacteriuria in cats.

The sub-aims of this study were to explore whether comorbidities previously associated with enterococcal bacteriuria including chronic kidney disease, hyperthyroidism, presence of a cystostomy tube, and spinal dysfunction (14) remained associated after performing a case-control analysis. This work was unable to confirm these diseases as risk factors for *Enterococcus* bacteriuria in cats, although the few patients with each of these pathologies limited our ability to make more definitive statements.

The study did have an additional interesting observation. Despite previous reports of feline enterococcal urinary tract infections displaying resistance to a greater number of antimicrobials than *E. coli* or other isolates, in this study there was not a difference between *Enterococcus* cases and *E. coli* controls (9,16). A potential explanation for why increased resistance was not seen in this study may be tied to the prevalence of *Enterococcus* species within our cohort. In this study, *E. faecalis* was the most common enterococci detected, comprising 74% of cases. *Enterococcus faecalis,* a commensal organism of the gastrointestinal tract, is the *Enterococcus* species most associated with virulence factors enabling the organism to induce urosepsis and invade human urothelial cells to create bacterial reservoirs (21–23). In contrast, *E. faecium* was isolated from only 10% of cat urine samples in this study. In humans, *E. faecium* has a higher incidence of antibiotic resistance compared with *E. faecalis* (24,25). It is postulated that increased resistance may be associated with a reduction in other aspects of fitness, including virulence (26). Thus, the increased virulence of *E. faecalis* may lead to a tradeoff of less bacterial resistance, and the predominance of *E. faecalis* in this study may explain the lack of a difference in resistance rates between *Enterococcus* cases and *E. coli* controls.

Finally, although outside our primary aim of investigation, signs of lower urinary tract disease (stranguria, dysuria, pollakiuria, and gross hematuria) were observed to be less common in cats with *Enterococcus* bacteriuria than control cats in this study, with 71% of cats with *Enterococcus* bacteriuria lacking associated clinical signs. This finding is in accordance with 2 previous studies noting an absence of clinical signs in approximately 55% of cats with *Enterococcus* bacteriuria (14,16). In addition, hematuria (on dipstick analysis) was less common in cats with *Enterococcus* bacteriuria than in cats with *E. coli* bacteriuria. These findings may suggest reduced pathogenicity of *Enterococcus* compared to *E. coli*. However, caution is warranted given that in cats, *Enterococcus* infections can be associated with pyelonephritis and bacteremia that may not be associated with overt signs of lower urinary tract disease (27,28).

The aim of this study was not to compare clinicopathologic variables between cats with *Enterococcus* bacteriuria with and

without clinical signs and therefore was not powered appropriately to make this determination. However, in this preliminary work, 11 of the 31 *Enterococcus* cases had microscopic hematuria. Of these cases, 4 had clinical signs and 7 had no clinical signs. Given that most of the urine samples were collected by cystocentesis, sampling-induced hematuria may be increasing hematuria detection particularly in the group of cats that lack clinical signs. Regardless, this observation may be clinically important since it is not known whether hematuria in the presence of bacteriuria is an indicator of symptomatic infection in cats. Similarly in humans, the current asymptomatic bacteriuria treatment guidelines do not discuss how to manage cases of hematuria and bacteriuria (29). In short, further studies comparing variables between cats with *Enterococcus* bacteriuria with and without clinical signs are warranted to identify parameters aside from clinical signs, such as the presence of hematuria on urinalysis, that may be associated with inflammation and disease.

The main limitations of this study were the small sample size and retrospective design. Missing values due to incomplete medical records further reduced the sample size for some but not all variables. In addition, this study evaluated the risk of developing enterococcal bacteriuria, and different risk factors may be associated with enterococcal UTI.

In conclusion, results of this retrospective study did not identify an association between factors causing urothelial damage and inflammation and enterococcal bacteriuria in cats as was hypothesized. When exploring other factors secondarily, we identified that cats with *Enterococcus* bacteriuria were less likely to have clinical signs and hematuria detected on urinalysis than cats with *E. coli* bacteriuria, and a comparison between cats with *Enterococcus* bacteriuria with and without clinical signs is necessary to identify parameters aside from clinical signs that may be associated with more severe disease. CVJ

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