PAPER

Risk factors for urinary bacterial growth in dogs with congenital portosystemic shunts: 66 cases (1997-2019)

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Objective: To identify risk factors for urinary bacterial growth in dogs with confirmed congenital portosystemic shunts on which a quantitative urine culture was performed.

Materials and Methods: Sixty-six dogs were included in this retrospective cross-sectional study. Medical records were reviewed from 1997 through 2019. Variables of interest included age, sex and sexual status, clinical signs for a urinary tract infection, blood urea concentration, urinalysis abnormalities, ultrasound abnormalities of the urinary tract, and previous treatment. Univariable and multivariable analyses were performed.

Results: The median age of the dogs was one year (range: 0.2-11.0 years). Urinary tract ultrasound abnormalities (cystic calculi and cystic debris) were reported in 50 dogs (75.7%). Abnormalities on urinalysis included pyuria in nine dogs (13.6%), bacteriuria in 13 dogs (19.7%), and haematuria in 26 dogs (39.4%). The median urine specific gravity was 1.021 (range: 1.004-1.052). Sixteen dogs (24.2%) had a positive quantitative urine culture. Based on multivariable analysis, bacteriuria (Odds ratio, 116; 95% CI, 9.6-1393; P = <0.001) was the only variable significantly associated with a significantly increased odds for a positive quantitative urine culture.

Clinical Significance: Clinical and subclinical bacteriuria can occur in dogs with congenital portosystemic shunts. In this group of dogs, bacteriuria was a risk factor for urinary bacterial growth.

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INTRODUCTION

Portosystemic shunts (PSSs) are vascular anomalies that connect blood flow from the portal vein to the systemic circulation, bypassing the hepatic parenchyma (Berent & Tobias 2009). PSSs can be congenital or acquired, and the latter are most often associated with chronic portal hypertension. Congenital portosystemic shunts (CPSSs) can be extrahepatic, which are more common in small breed dogs, or intrahepatic, which are more common in large breed dogs (Payne *et al.* 1990, Lamb & White 1998). Dogs with PSSs can have a variety of clinical abnormalities. These include retarded growth, coagulopathies and gastrointestinal, neurologic, and/or lower urinary tract signs (Winkler *et al.* 2003, Kraun *et al.* 2014, Lidbury *et al.* 2015). In the literature, common urinary signs include haematuria, pollakiuria, stranguria, polyuria, polydipsia, presence of urate calculi, and bacterial growth and/or infection (Winkler *et al.* 2003, Kraun *et al.* 2014). Urate urolithiasis is attributed to decreased urea production, decreased uric acid metabolism, and increased ammonia excretion (Bartges *et al.* 1999, Winkler *et al.* 2003, Kraun *et al.* 2014, Eh *et al.* 2015).

It is widely reported that the common risk factors for developing urinary bacterial growth (UBG) and subsequent urinary tract infection (UTI) include female sex, urolithiasis, and anatomical or functional abnormalities of the urinary tract (Byron 2019). Several veterinary studies reported that diseases such as diabetes mellitus, hyperadrenocorticism, thoracolumbar vertebral disease, and chronic kidney disease (CKD) have been associated with an increased risk for developing UBG and UTI (Forrester *et al.* 1999, Olby *et al.* 2010, Lamoureux

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et al. 2019). Although UBG/UTI have been reported in dogs with CPSSs, risk factors for this population still remain undetermined. One study suggested that the presence of urate calculi could result in secondary bacterial tract infections (Winkler *et al.* 2003).

The main objective of this study was to describe and identify risk factors for UBG in dogs with CPSSs at the time of clinical and diagnostic evaluation. We hypothesized that dogs with CPSSs with evidence of certain lower urinary tract abnormalities will have an increased risk for developing UBG. A literature search was performed using databases such as PubMed, MEDLINE, ClinicalKey, Biosys and Agricola on October 6, 2020. The following keywords were used for this search: dogs, congenital portosystemic shunts, UTIs, risk factors, and subclinical bacteriuria (SUB). Based on this search, no reports describing risk factors for UBG in dogs with CPSSs were found.

MATERIALS AND METHODS

Medical record search

Electronic medical records were searched from 1997 through 2019 to identify dogs with a diagnosis for CPPSs. We searched for the following keywords: congenital portosystemic shunts, portosystemic shunts, and intra-and extrahepatic portosystemic shunts.

Inclusion criteria

Inclusion criteria included the following: (1) evidence of a single intra or extra-hepatic anomalous vessel that provided direct vascular communication between the portal venous supply and the systemic venous circulation diagnosed by abdominal ultrasound (AUS), scintigraphy, CT, MRI, or exploratory laparotomy; (2) a record for a complete AUS with a detailed description of the urinary tract; (3) records that at least one quantitative urinary culture (QUC) was performed within 48 hours of CPSSs diagnosis with sterile urine obtained by cystocentesis along with a minimum database diagnostic lab work (complete blood count, biochemistry profile, and urinalysis); (4) records showing that the inclusion visit was before shunt attenuation as all dogs undergoing surgery would have had received antibiotic therapy therefore, modifying QUC results.

Exclusion criteria

Exclusion criteria included dogs that did not fulfil the criteria listed above or received antimicrobial treatment within the 30 days before urinary bacterial culture. Patients with evidence of multiple shunts were also excluded. Treatment with neomycin and/or metronidazole was considered an exemption as these drugs are known to mainly act within the gastrointestinal tract and although metronidazole metabolites can be excreted in the urine, these are relatively ineffective against facultative aerobic bacteria, which are commonly associated with UTIs in dogs (Plumb 2015, Dingsdag & Hunter 2018). Also, patients with evidence of another underlying disease were excluded.

Data collection

Medical records from each dog were reviewed and the following data were obtained: age, sex and sexual status, lower urinary tract signs (*e.g.* gross haematuria, dysuria, pollakiuria, or stranguria), evidence of lower urinary signs reported by the owner and/or the attending clinician, shunt type (*i.e.* extrahepatic or intrahepatic), ultrasonographic urinary tract abnormalities (*i.e.* presence of cystic and/or renal calculi, and cystic echogenic debris suggestive of cystitis), evidence of low BUN on serum biochemistry panel values, and presence or absence of abnormalities on urinalysis (*e.g.* pyuria, haematuria, bacteriuria, pH, and specific gravity).

Medical records indicated that BUN measurements (Vitros 4600 Chemistry System, Ortho-Clinical Diagnostics, Raritan, NJ) were performed by in house analysers following standard operating procedures. Urine was routinely collected through cystocentesis, and was analysed. Urine specific gravity (USG) was measured via refractometry and a fresh multistick 10SG (Siemens, Germany) was used for chemical analysis. The multistick was dipped into a Kova Tube (Kova international, CA, USA) with urine, patted dry on its side with a gauze and placed on a Clinitek Urinalysis Analyzer (Siemens, Germany) for evaluation. Urinary sediment analysis was performed by a laboratory technician. Pyuria was defined as more than five leukocytes per high power field (HPF) and microscopic haematuria was defined as >10 erythrocytes/HPF. The presence of bacteria was confirmed using a modified Wright-Giemsa stain. Urine was also submitted for QUC. Samples were cultured in two blood agar plates (BAP) and one MacConkey (MAC) agar plate. For the BAP plates, two dilutions were used: 1:100 and 1:1000, respectively. Incubation was performed in 6.1% CO_2 at 37°C for BAP plates and at 37°C in an air incubator for MAC agar plate. A positive result was considered when ≥1000 CFU/mL were observed.

Measured outcomes

The a priori outcome measurement was the presence or absence of UBG, confirmed by QUC.

Statistical analyses

Continuous data (age and USG) were transformed to normal quantile plots and normality was assessed using a Shapiro-Wilk W Test. Median and range are provided for continuous data. Initially age was analysed as a continuous variable, but no significant association was identified. Therefore, it was analysed as a categorical variable forming two groups according to the obtained median (juvenile: <1 year or adult: >1 year). All categorical data were expressed in percentages and included: age (juvenile or adult), sex and sexual status (female spayed, male castrated, male intact, or female intact), lower urinary tract signs (present or absent), type of lower urinary tract sign (gross haematuria, pollakiuria, and dysuria), presence of a low BUN (yes or no), evidence of echogenic debris suggestive of cystitis on AUS (yes or no), evidence of urolithiasis on AUS (yes or no), urine pH (<6.0, 6.0-7.5, or >7.5), presence of haematuria (yes or no), pyuria (yes or no) or bacteriuria (yes or no) on urinalysis, shunt type (extrahepatic or intrahepatic), and previous treatment (metronidazole, neomycin, or none).

The only variable of interest from the patients' biochemistry panels was the presence or absence of BUN concentration below the lower limit of the reference interval. The justification for this is based on the fact that low BUN concentrations due to impaired hepatic urea cycle activity can affect the renal medullary concentration gradient and may contribute to low urine concentration (Deppe *et al.* 1999). Several reports may suggest that low urine concentration can be a risk factor for developing urinary bacterial overgrowth (Puchot *et al.* 2017, Lamoureux *et al.* 2019).

Univariable analysis was performed using Fisher's exact or Chi square tests for categorical variables. The outcome variable used was positive UBG at initial diagnosis for CPSSs. Explanatory variables (putative risk factors) evaluated included age, sex and sexual status, lower urinary tract signs, type of lower urinary tract sign, low BUN, ultrasound abnormalities, urinalysis abnormalities, shunt type, and previous antibiotic treatment. Since urinary pH had more than two categories, the category corresponding to normal values (6.5-7.5) was used as the reference category. Results are reported as odds ratios (ORs) with their 95% confidence intervals (CIs). Variables with a P value <0.2 on the univariable analysis were included in a multivariable logistic regression model and backward stepwise elimination was used to determine a final model. All differences were considered significant if P values were ≤0.05. Statistical analysis was carried out using a commercially available statistical software (JMP Version 15. SAS Institute Inc., Cary, NC).

RESULTS

Descriptive statistics

We screened a total of 594 records and identified 66 dogs that fulfilled the inclusion criteria (Fig 1). The median age of the 66 dogs at the time of diagnosis was one year (range: min: 0.2 years, max: 11.0 years). Thirty-five dogs were <1 year of age (53%) and 31 were >1 year of age (47%). There were 33 spayed females (50%), 15 castrated males (23%), 12 intact males (18%) and six intact females (9%). Twenty-six breeds were represented: 15 Yorkshire Terriers (2.7%), seven Miniature Schnauzers (10.6%), five Standard Schnauzers (7.5%), two Jack Russell Terriers (3%), three Maltese (4.5%), three mixed breed dogs (4.5%), three Miniature Poodles (4.5%), three Shih-tzu (4.5%), two Bichon Frise (3%), two Chihuahuas (3%), two Golden Retrievers (3%), two Irish Wolfhounds (3%), two Labrador Retrievers (3%), two Pugs (3%), and two Standard Poodles (3%). In addition, there was one (1.5%) of each of the following breeds: Australian Heeler, Boxer, Cocker Spaniel, Collie, Dachshund, Doberman Pinscher, Miniature Pinscher, Nova Scotia Duck Trolling Retriever, Pembroke Welsh Corgi, Rhodesian Ridgeback, and West Highland White Terrier.

Thirteen dogs (19.7%) had a history of receiving antibiotic treatment with metronidazole (7 dogs [10.7%]) or neomycin (6 dogs [9%]).

Fifty-three dogs were diagnosed with extrahepatic portosystemic shunts (80.3%) and 13 dogs with intrahepatic portosystemic shunts (19.7%). The most common diagnostic method for CPSSs was AUS in 61 dogs (92.4%) followed by scintigraphy in five dogs (7.6%).

Only 21 dogs (31.8%) had a history of lower urinary tract signs and 45 dogs (68.2%) had no clinical signs. Gross haematuria was the most common, reported in 12 dogs (18.2%), followed by stranguria in eight dogs (12.2%), and lastly pollakiuria in one dog (1.5%).

Urinary tract abnormalities reported by AUS included: cystic calculi in 43 dogs (65.2%) and echogenic debris suggestive of cystitis in 21 dogs (31.8%).

Serum BUN concentration was measured in all 66 dogs. Low BUN was reported in 19 dogs (28.8%). A urinalysis was

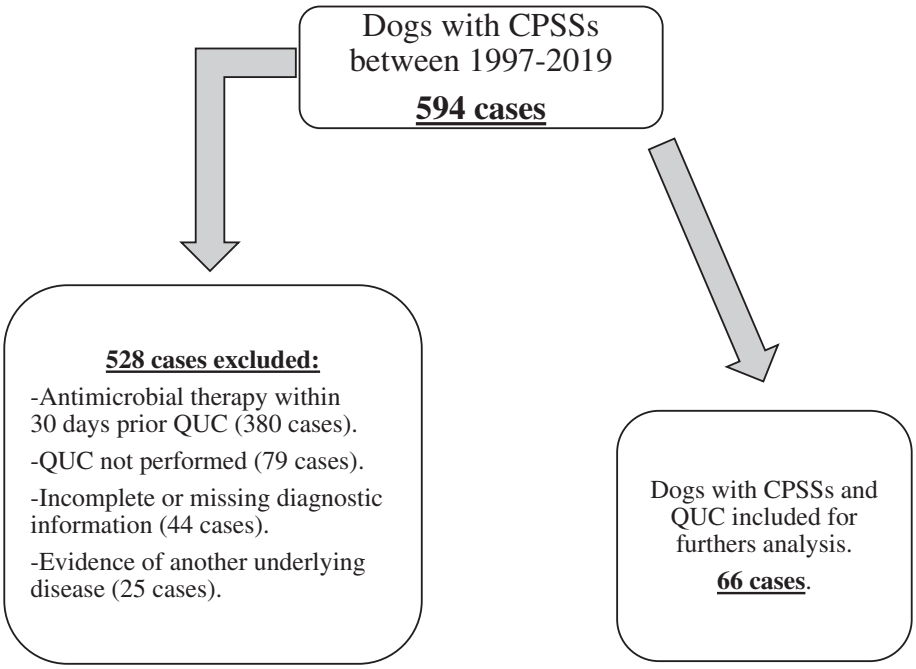

FIG 1. Case selection process. CPSSs congenital portosystemic shunts, QUC quantitative urine culture

performed in all dogs. Nine dogs (13.6%) had pyuria, 26 dogs (39.4%) had haematuria, and 13 dogs (19.7%) had bacteriuria. Thirty-three dogs (50%) had an inactive urinary sediment. The median pH for all dogs was 7 (min: 5.5, max: 8.5), and the median specific gravity was 1.021 (min: 1.004, max: 1.052).

A positive QUC was reported in 16 dogs (24.2%). Isolated bacteria included: *Escherichia coli* in eight dogs (50%), *Pseudomonas aeruginosa* in three dogs (19%), *Staphylococcus intermedius* in three dogs (19%), and *Proteus mirabilis* and *Enterococcus* spp. were present in one dog (6%) each. From the positive QUC group, only six dogs (37.5%) were reported to have lower urinary tract signs, with three dogs (18.7%) presenting with gross haematuria and stranguria, respectively. Nine dogs were spayed females (56.3%), three dogs were castrated males (18.7%), two dogs were intact males (12.5%), and two dogs were intact females (12.5%). Reported ages for this subgroup were 11 dogs >1 year of age (68.7%) and five dogs <1 year of age (31.3%).

Risk factor analysis

Results obtained from the univariable and multivariable analyses are summarised in Table 1. Age, sex and sexual status, presence of cystic calculi, echogenic debris suggestive of cystitis, low BUN, shunt type, presence and type of lower urinary tract signs, presence of haematuria, USG, and previous treatment with metronidazole or neomycin were not associated with UBG at the time of evaluation on univariable analysis. However, the presence of bacteriuria was significantly associated with UBG (OR, 147; 95% CI, 15-1437; P = <0.001). Similarly, the presence of pyuria was significantly associated with UBG (OR, 5.2; 95% CI, 1.2-22, P = 0.031). When pH values were grouped according to reference intervals (Piech & Wycislo 2019), dogs with a urine pH >7.5 were significantly associated with UBG (OR, 6.7; 95% CI, 1.9-23; P = 0.002). Pyuria, USG, shunt type (intrahepatic *versus* extrahepatic) low BUN, bacteriuria, and urine pH >7.5 were included in the multivariate analysis (P<0.2 on univariable analysis; Table 1). After backward stepwise elimination, only bacteriuria (OR, 116; 95% CI, 9.9-1393; P = <0.001) remained in the final model.

DISCUSSION

The frequency of UBG in 66 dogs with CPSSs for which a QUC was performed was 24.2%, but of these only 37.5% had lower urinary tract signs. These results are similar to those for other diseases such as CKD (Lamoureux *et al.* 2019), intervertebral

TABLE 1. Univariable and multivariable logistic regression analyses to evaluate risk factors for urinary bacterial growth in 66 dogs with diagnosed CPSSs

		Univariable analyses		Multivariable analyses	
Factor	**No of Dogs**	**OR (95% CIs)**	**P value**	**OR (95% CIs)**	**P value**
Age (y)					
Juvenile (<1)	35	2.3 (0.7-7.3)	0.24		
Adult (>1)	31				
Sex and sexual status					
Female spayed	33	1.2 (0.1-9)	0.80	–	–
Male castrated	15	0.8 (0.1-5.7)	0.52		
Female intact	12	0.4 (0.0-3.9)	0.52		
Male intact	6				
Lower urinary tract signs					
Yes	21	1.4 (0.4-4.5)	0.39	–	–
No	45				
Lower urinary tract sign					
Gross haematuria	12	1.8 (0.2-12)	0.55	–	–
Stranguria	8	0.4 (0.1-2.3)	0.99		
Low BUN					
Yes	19	2.5 (0.7-8)	**0.11**	1.2 (0.13-10)	0.87
No	47				
AUS abnormalities					
Cystic calculi	43	1.2 (0.3–4.1)	0.48	–	–
Echogenic debris	21	1.5 (0.4-5.5)	0.83	–	–
Urinalysis					
Haematuria	26	1.7 (0.6-5.5)	0.23		
Pyuria	9	5.2 (1.2-22)	**0.031**	2.4 (0.1-37)	0.44
Bacteriuria	13	147.0 (15-1437)	**<0.001**	116.0 (9.6-1393)	**<0.001**
USG	66	1.6 (0.0-5.6)	**0.07**	1.7 (0.0-162)	0.71
pH (>7.5)	17	6.7 (1.9-23)	**0.002**	5 (0.6-40)	0.055
Shunt type					
Intrahepatic	13	2.3 (0.6-8)	**0.16**	1.3 (0.1-13)	0.80
Extrahepatic	53	0.4 (0.1-1.5)	0.95	–	–
Previous treatment					
Metronidazole	7	0.8 (0.14-4.6)	0.65	–	–
Neomycin	6	0.5 (0.03-4.7)	0.61	–	–

Univariable analysis was performed using Fisher's exact or Chi square tests. Variables with a P value <0.2 on the univariable analysis (on bold) were included in a multivariable logistic regression model. The results of the initial multivariable model (before backward stepwise elimination) are reported here
AUS Abdominal ultrasound, BUN Blood urea nitrogen, CIs Confidence intervals, OR Odds ratio, USG Urine specific gravity, UTI urinary tract infection

disc disease (Olby *et al*. 2010), hyperadrenocorticism (Forrester *et al*. 1999), and diabetes mellitus (Forrester *et al*. 1999). Furthermore *E. coli* was the most frequently isolated bacteria in this group, accounting for 50% of the reported isolates. This is no surprise as it is well documented in humans and dogs that non-gastrointestinal *E. coli* possess extraintestinal virulence factors, making them more likely to become uropathogens. These factors include the production of siderophores, cytotoxins, transport systems and expression of adhesins that mediate binding to the renal tubule (P, S, and F1C fimbriae), and urothelium (type 1 fimbriae), facilitating adherence and invasion to these biological surfaces (Thompson *et al*. 2011, Olin & Bartges 2015, Byron 2019). Previous reports showed that spayed female dogs are overrepresented for developing UBG, this is mainly attributed to anatomical differences (Hall *et al*. 2013, Wan *et al*. 2014). We observed that sex was not a risk factor for developing UBG in this population: although 68.75% of dogs with UBG were female a considerable number of female dogs also had a negative QUC. This difference in findings may be due to differences in populations between studies and the relatively small sample size in our study.

Our study showed that bacteriuria was a significant risk factor for a positive QUC. This finding was expected as it has been previously observed in dogs and cats (Puchot *et al*. 2017, Rafatpanah Baigi *et al*. 2017, Grimes *et al*. 2020). Three dogs (18.8%) with a positive QUC had a negative sediment. Of these three dogs, only one dog (33%) had no signs of lower urinary tract disease. The combination of bacteriuria and pyuria leads to a high suspicion of UTI and it is used in people as a screening tool to assess the need for culture, therefore, an adequate microscopic sediment examination is paramount (Kass & Finland 2002, Nicolle *et al*. 2005). Several studies demonstrated that Gram staining and Wright-Giemsa staining on wet mount and air-dried preparations will have a higher specificity and sensitivity for detecting bacteriuria in dogs and cats (Swenson *et al*. 2011, O'Neil *et al*. 2013). Pyuria alone can be an inconsistent finding in dogs with bacteriuria and thus should not be relied upon as an indicator of an absence of infection (Swenson *et al*. 2011, Puchot *et al*. 2017). This inconsistency was also observed in our study as only 31% of dogs with pyuria had a positive QUC and although pyuria was significant on univariable analysis, this association was not significant on multivariable analysis, possibly due to a confounding factor.

In this study population, the median urinary pH was seven. It is known that large amounts of ammonia in the urine, tend to alkalinize urinary pH, although when in combination with uric acid metabolites, urinary pH tends be more acidic and even be a predisposing factor for lithiogenesis (Bartges *et al*. 1999, Bartges & CallenS 2015, Eh *et al*. 2015). This could represent a possible explanation for this finding as 43 dogs (65%) in this study were reported to have urolithiasis. Interestingly, our study showed significance (P = 0.002) on univariable analysis, but was not significant (P = 0.055) on multivariate analysis that dogs with an alkaline urine (pH > 7.5) had higher odds of having UBG compared to dogs with normal urine pH. As pH did not reach the threshold for statistical significance on multivariable analysis, this association is questionable. Regardless, our cross-sectional study design could not allow determination of cause and effect and so it is possible the alkaline urine occurred as a result of UBG or that alkaline urine pH predisposed dogs with CPSSs to UBG. Prior work supports either possibility depending on the species of bacteria involved. Alkaline urine can occur due to the presence of the urease-positive organisms *Proteus spp., Staphylococci*, or *Corynebacterium urealyticum* (Stockham & Scott 2008). In our study, five dogs (31%) with a positive QUC had one of these organisms isolated. Also, when assessing alkaline urine pH, it is important to remember that alkaline urine occurs physiologically for several hours postprandially, which is also known as the alkaline tide (Stockham & Scott 2008). Due to the retrospective nature of this study, we were not aware of the prandial status for all dogs. Although, it is worth pointing out that sample collection protocols at our teaching hospital requires a fasting period of at least 8 hours for all dogs before any blood samples for complete blood count and chemistry panel are withdrawn. Certain urine physicochemical properties such as the low pH and high osmolality may serve as defence mechanisms against bacterial colonisation in the lower urinary tract (Smee *et al*. 2013). Although some human studies have shown that acidic urine and increased osmolality can inhibit *E. coli* growth *in vitro* (Kaye 1968), these findings have been inconsistent in veterinary research (Forrester *et al*. 1999, Bailiff *et al*. 2006). A previous study showed that *E. coli in vitro* growth was higher in neutral to acidic and diluted urine compared to alkaline and concentrated urine from healthy dogs (Thornton *et al*. 2018).

We reported that 10 dogs (62.5%) with a positive QUC had absence of lower urinary tract signs. SUB is defined as the presence of bacteria in the urine in the absence of clinical signs and confirmed with a positive QUC (Weese *et al*. 2019). The rates for SUB reported here are similar to other reports. These studies mainly investigated the prevalence of SUB in groups of dogs that underwent a surgical procedure, or presented with a chronic illness (Olby *et al*. 2010, McGhie *et al*. 2014, Rafatpanah Baigi *et al*. 2017). Polyuria/polydipsia (PU/PD) is common in dogs with CPSSs, leading to low USG. This likely results from poor medullary concentration gradient due to low BUN levels, increased renal blood flow and psychogenic polydipsia (Berent & Tobias 2009). Low USG had been suggested to be a cause of inactive urine sediment in dogs with UBG (Tivapasi *et al*. 2009). We found that in this population of dogs the median USG was 1.021, and although this value may question the severity of PU/PD, it is well under the threshold for minimal concentrating ability (1.030), and similar to previous reports in dogs with CPSSs (Deppe *et al*. 1999, Kraun *et al*. 2014, Eh *et al*. 2015). Therefore, it is possible that low USG could explain some of the discordant cases. As previously mentioned a high USG is associated with high presence of antimicrobial factors thus, low urine concentration could be an explanation for SUB as similar studies in dogs and cats with other comorbidities suggest (Forrester *et al*. 1999, Puchot *et al*. 2017, Lamoureux *et al*. 2019). It has been proposed in human medicine that certain strains of *E. coli* lack virulence factors and could be associated with SUB (Vejborg *et al*. 2012). Although this has not been demonstrated in dogs, it could represent a possible explanation given that *E. coli* was the most com-

mon isolated pathogen in our study and others (Wan *et al.* 2014, Foster *et al.* 2018, Lamoureux *et al.* 2019). Another important aspect to consider was the retrospective nature of this study, as it cannot be ruled out that some of the enrolled dogs had clinical signs that were however not noticed or not recorded.

Historically, it has been reported that the development of ammonium urate stones and subsequent cystitis can represent a risk factor for developing a UTI (Winkler *et al.* 2003, Berent & Tobias 2009). Our study found no association between urolithiasis and UBG. It has been observed that dogs with urolithiasis will present many times with a negative QUC due to host defence mechanisms. In these situations, a mucosal biopsy of the bladder and urolith cultures are recommended to accurately assess the microbiological status of the urinary tract (Gatoria *et al.* 2006). These procedures were not performed in our study.

Limitations of this study include its retrospective design that could potentially lead to inaccurate information in each dog's records, especially when reporting signs of lower urinary tract disease, the observance of which could vary between owners and clinicians. We were also unable to determine the prevalence of UBG in dogs with CPSSs due to the possibility of selection *i.e.* many dogs that were excluded did not have a QUC, possibly due to the clinician's decision in the absence of lower urinary tract signs. However, 62.5% of the dogs with UBG did not have lower urinary tract signs recorded. Finally, it is possible that the risk factor analysis was underpowered due to the relatively small sample size. Additionally, there is a considerable likelihood that a type 2 error could be present in our analyses, as only 16 dogs were reported to have a positive urine culture in our study.

In this group of dogs with CPSSs, the prevalence for UBG was 24.2%, which was apparently subclinical in 62.5% of the cases. Bacteriuria was the only variable associated with UBG on multivariate analysis. Prospective studies are needed to further determine the prevalence of and the risk factors for UBG in dogs with CPSSs.

Conflict of interest

No conflicts of interest have been declared.

References

Bailiff, N. L., Nelson, R. W., Feldman, E. C., *et al*. (2006) Frequency and risk factors for urinary tract infection in cats with diabetes mellitus. *Journal of Veterinary Internal Medicine* **20**, 850-855

Bartges, J. W. & CallenS, A. J. (2015) Urolithiasis. *The Veterinary Clinics of North America. Small Animal Practice* **45**, 747-768

Bartges, J. W., Osborne, C. A., Lulich, J. P., *et al*. (1999) Canine urate urolithiasis. Etiopathogenesis, diagnosis, and management. *The Veterinary Clinics of North America. Small Animal Practice* **29**, 161-191, xii-xiii

Berent, A. C. & Tobias, K. M. (2009) Portosystemic vascular anomalies. *The Veterinary Clinics of North America. Small Animal Practice* **39**, 513-541

Byron, J. K. (2019) Urinary tract infection. *The Veterinary Clinics of North America. Small Animal Practice* **49**, 211-221

Deppe, T. A., Center, S. A., Simpson, K. W., *et al*. (1999) Glomerular filtration rate and renal volume in dogs with congenital portosystemic vascular anomalies before and after surgical ligation. *Journal of Veterinary Internal Medicine* **13**, 465-471

Dingsdag, S. A. & Hunter, N. (2018) Metronidazole: an update on metabolism, structure-cytotoxicity and resistance mechanisms. *The Journal of Antimicrobial Chemotherapy* **73**, 265-279

Eh, G. C., Phillips, H., Underwood, L., *et al*. (2015) Risk factors for urolithiasis in dogs with congenital extrahepatic portosystemic shunts: 95 cases (1999-2013). *Journal of the American Veterinary Medical Association* **246**, 530-536

Forrester, S. D., Troy, G. C., Dalton, M. N., *et al*. (1999) Retrospective evaluation of urinary tract infection in 42 dogs with hyperadrenocorticism or diabetes mellitus or both. *Journal of Veterinary Internal Medicine* **13**, 557-560

Foster, J. D., Krishnan, H. & Cole, S. (2018) Characterization of subclinical bacteriuria, bacterial cystitis, and pyelonephritis in dogs with chronic kidney disease. *Journal of the American Veterinary Medical Association* **252**, 1257-1262

Gatoria, I. S., Saini, N. S., Rai, T. S., *et al*. (2006) Comparison of three techniques for the diagnosis of urinary tract infections in dogs with urolithiasis. *The Journal of Small Animal Practice* **47**, 727-732

Grimes, M., Heseltine, J. C., Nabity, M. B., *et al*. (2020) Characteristics associated with bacterial growth in urine in 451 proteinuric dogs (2008-2018). *Journal of Veterinary Internal Medicine* **34**, 770-776

Hall, J. L., Holmes, M. A. & Baines, S. J. (2013) Prevalence and antimicrobial resistance of canine urinary tract pathogens. *The Veterinary Record* **173**, 549

Kass, E. H. & Finland, M. (2002) Asymptomatic infections of the urinary tract. *The Journal of Urology* **168**, 420-424

Kaye, D. (1968) Antibacterial activity of human urine. *The Journal of Clinical Investigation* **47**, 2374-2390

Kraun, M. B., Nelson, L. L., Hauptman, J. G., *et al*. (2014) Analysis of the relationship of extrahepatic portosystemic shunt morphology with clinical variables in dogs: 53 cases (2009-2012). *Journal of the American Veterinary Medical Association* **245**, 540-549

Lamb, C. R. & White, R. N. (1998) Morphology of congenital intrahepatic portacaval shunts in dogs and cats. *The Veterinary Record* **142**, 55-60

Lamoureux, A., Da Riz, F., Cappelle, J., *et al*. (2019) Frequency of bacteriuria in dogs with chronic kidney disease: a retrospective study of 201 cases. *Journal of Veterinary Internal Medicine* **33**, 640-647

Lidbury, J. A., Ivanek, R., Suchodolski, J. S., *et al*. (2015) Putative precipitating factors for hepatic encephalopathy in dogs: 118 cases (1991-2014). *Journal of the American Veterinary Medical Association* **247**, 176-183

McGhie, J. A., Stayt, J. & Hosgood, G. L. (2014) Prevalence of bacteriuria in dogs without clinical signs of urinary tract infection presenting for elective surgical procedures. *Australian Veterinary Journal* **92**, 33-37

Nicolle, L. E., Bradley, S., Colgan, R., *et al*. (2005) Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. *Clinical Infectious Diseases* **40**, 643-654

Olby, N. J., Mackillop, E., Cerda-Gonzalez, S., *et al*. (2010) Prevalence of urinary tract infection in dogs after surgery for thoracolumbar intervertebral disc extrusion. *Journal of Veterinary Internal Medicine* **24**, 1106-1111

Olin, S. J. & Bartges, J. W. (2015) Urinary tract infections: treatment/comparative therapeutics. *The Veterinary Clinics of North America. Small Animal Practice* **45**, 721-746

O'Neil, E., Horney, B., Burton, S., *et al*. (2013) Comparison of wet-mount, Wright-Giemsa and gram-stained urine sediment for predicting bacteriuria in dogs and cats. *The Canadian Veterinary Journal* **54**, 1061-1066

Payne, J. T., Martin, R. A. & Constantinescu, G. M. (1990) The anatomy and embryology of portosystemic shunts in dogs and cats. *Seminars in Veterinary Medicine and Surgery (Small Animal)* **5**, 76-82

Piech, T. L. & Wycislo, K. L. (2019) Importance of urinalysis. *The Veterinary Clinics of North America. Small Animal Practice* **49**, 233-245

Plumb, D. C. (2015) Plumb's Veterinary Drug Handbook. Wiley-Blackwell, Ames, IA, USA

Puchot, M. L., Cook, A. K. & Pohlit, C. (2017) Subclinical bacteriuria in cats: prevalence, findings on contemporaneous urinalyses and clinical risk factors. *Journal of Feline Medicine and Surgery* **19**, 1238-1244

Rafatpanah Baigi, S., Vaden, S. & Olby, N. J. (2017) The frequency and clinical implications of bacteriuria in chronically paralyzed dogs. *Journal of Veterinary Internal Medicine* **31**, 1790-1795

Smee, N., Loyd, K. & Grauer, G. (2013) UTIs in small animal patients: part 1: etiology and pathogenesis. *Journal of the American Animal Hospital Association* **49**, 1-7

Stockham, S. L. & Scott, M. A. (2008) Fundamentals of Veterinary Clinical Pathology. Ames, IA, USA, Blackwell Pub

Swenson, C. L., Boisvert, A. M., Gibbons-Burgener, S. N., *et al*. (2011) Evaluation of modified Wright-staining of dried urinary sediment as a method for accurate detection of bacteriuria in cats. *Veterinary Clinical Pathology* **40**, 256-264

Thompson, M. F., Litster, A. L., Platell, J. L., *et al*. (2011) Canine bacterial urinary tract infections: new developments in old pathogens. *Veterinary Journal* **190**, 22-27

Thornton, L. A., Burchell, R. K., Burton, S. E., *et al*. (2018) The effect of urine concentration and pH on the growth of *Escherichia coli* in canine urine in vitro. *Journal of Veterinary Internal Medicine* **32**, 752-756

Tivapasi, M. T., Hodges, J., Byrne, B. A., *et al*. (2009) Diagnostic utility and cost-effectiveness of reflex bacterial culture for the detection of urinary tract infection in dogs with low urine specific gravity. *Veterinary Clinical Pathology* **38**, 337-342

Vejborg, R. M., De Evgrafov, M. R., Phan, M. D., *et al*. (2012) Identification of genes important for growth of asymptomatic bacteriuria *Escherichia coli* in urine. *Infection and Immunity* **80**, 3179-3188

Wan, S. Y., Hartmann, F. A., Jooss, M. K., *et al*. (2014) Prevalence and clinical outcome of subclinical bacteriuria in female dogs. *Journal of the American Veterinary Medical Association* **245**, 106-112

Weese, J. S., Blondeau, J., Boothe, D., *et al*. (2019) International Society for Companion Animal Infectious Diseases (ISCAID) guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats. *Veterinary Journal* **247**, 8-25

Winkler, J. T., Bohling, M. W., Tillson, D. M., *et al*. (2003) Portosystemic shunts: diagnosis, prognosis, and treatment of 64 cases (1993-2001). *Journal of the American Animal Hospital Association* **39**, 169-185