Frequency of urinary tract infection among dogs with pruritic disorders receiving long-term glucocorticoid treatment

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Objective—To determine frequency of urinary tract infection (UTI) among dogs with pruritic disorders that were or were not receiving long-term glucocorticoid treatment.

Design—Observational study.

Animals—127 dogs receiving glucocorticoids for > 6 months and 94 dogs not receiving glucocorticoids.

Procedure—Bacterial culture of urine samples was performed in dogs receiving long-term glucocorticoid treatment, and information was collected on drug administered, dosage, frequency of administration, duration of glucocorticoid treatment, and clinical signs of UTI. For dogs not receiving glucocorticoids, a single urine sample was submitted for bacterial culture.

Results—Multiple (2 to 6) urine samples were submitted for 70 of the 127 (55%) dogs receiving glucocorticoids; thus, 240 urine samples were analyzed. For 23 of the 127 (18.1%) dogs, results of bacterial culture were positive at least once, but none of the dogs had clinical signs of UTI. Pyuria and bacteriuria (present vs absent) were found to correctly predict results of bacterial culture for 89.9% and 95.8% of the samples, respectively. Type of glycocorticoid, dosage, frequency of administration, and duration of treatment were not associated with frequency of UTI. None of the urine samples from dogs not receiving glucocorticoids yielded bacterial growth. The frequency of UTI was significantly higher for dogs treated with glucocorticoids than for dogs that had not received glucocorticoids.

Conclusions and Clinical Relevance—Results suggest that dogs receiving long-term glucocorticoid treatment have an increased risk of developing a UTI. On this basis, we recommend that urine samples be submitted for bacterial culture at least yearly for such dogs. (*J Am Vet Med Assoc* 2005;227:239–243)

Glucocorticoids are among the most commonly used medications in veterinary medicine[1-3] and are currently the most affordable and most efficacious anti-inflammatory drugs for treating dogs with chronic pruritic skin disorders. Unfortunately, long-term use of glucocorticoids can be associated with multiple adverse effects.[4-6]

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Several authors have suggested that glucocorticoid treatment, by suppressing systemic and local immune responses, may predispose dogs to develop **urinary tract infections (UTIs)**.[3,4,7] However, to our knowledge, only a single clinical study[3] supporting this view has been published. In that study, 39% of dogs with various inflammatory disorders that received long-term glucocorticoid treatment developed a UTI. Providing additional support for the suggestion that glucocorticoids predispose dogs to develop UTIs is the finding in a previous study[8] that 46% of dogs with hyperadrenocorticism had a UTI.

Dogs that develop UTIs during glucocorticoid treatment may have minimal or no clinical signs, and urine samples from these dogs may not have evidence of pyuria or bacteriuria.[3] Therefore, bacterial culture of urine samples may be necessary to identify UTI in some dogs receiving long-term glucocorticoid treatment. Failure to recognize and treat UTIs in these dogs may result in serious consequences, such as pyelonephritis.[9,10]

The purpose of the study reported here was to determine whether the frequency of UTI among dogs with pruritic disorders that received long-term oral glucocorticoid treatment was significantly greater than the frequency among dogs with pruritic disorders that did not receive glucocorticoid treatment. In addition, we sought to determine whether urinalysis was a satisfactory test for UTI in dogs receiving long-term glucocorticoid treatment. Finally, we wanted to determine whether type of glucocorticoid, dosage, frequency of glucocorticoid administration, or duration of treatment was associated with frequency of UTI in dogs receiving long-term glucocorticoid administration.

Materials and Methods

Dogs—To determine the frequency of UTI among dogs with pruritic disorders that received long-term oral glucocorticoid treatment, medical records of dogs treated by the University of Minnesota Veterinary Dermatology Service between 1994 and 2003 were reviewed. Dogs of any age, sex, or breed that had a pruritic disorder and received long-term glucocorticoid treatment were eligible for inclusion. Dogs were included only if at least 1 urine sample obtained at least 6 months after initiation of glucocorticoid treatment had been submitted for bacterial culture. Dogs that received glucocorticoid treatment for < 6 months were excluded. Additionally, information was recorded only for urine samples obtained by means of cystocentesis. Information obtained from the medical records for dogs included in the study consisted of breed, age, sex, neuter status, type of glucocorticoid administered, dosage, frequency of administra-

tion, duration of treatment, results of urinalysis, results of bacterial culture of urine samples and antimicrobial susceptibility testing, interval between submission of urine samples, and clinical signs of UTI.

To determine the frequency of UTI among dogs with pruritic disorders that did not receive glucocorticoid treatment, dogs treated by the University of Minnesota Veterinary Dermatology Service between June 2003 and July 2004 were enrolled in the study. Dogs of any age, sex, or breed that had a pruritic disorder and had not received long-term glucocorticoid treatment were eligible for inclusion in the study. Dogs that had received glucocorticoids topically or systemically during the preceding 6 months were excluded, as were dogs that had received glucocorticoids for > 4 weeks at any time in the past. Client consent was obtained before dogs were enrolled in the study. The study protocol was approved by the University of Minnesota Institutional Animal Care and Use Committee.

For dogs enrolled in the study, a urine sample was obtained by means of cystocentesis and submitted for bacterial culture. Breed, age, sex, neuter status, and results of bacterial culture of the urine sample and antimicrobial susceptibility testing were recorded. Owners were specifically asked whether their dogs had any clinical signs of UTI.

Bacterial culture and susceptibility testing—Urine samples were plated on blood agar and MacConkey agar plates, and bacteria were identified on the basis of macroscopic colony characteristics and results of standard biochemical tests. Dogs were considered to have a UTI if bacterial culture yielded > 1,000 colony-forming units/mL of urine. Antimicrobial susceptibility was determined with a commercially available kit.[a]

Urine sediment analysis—A drop of mixed urine sediment was placed on a microscope slide and covered with a coverslip. Formed elements, with the exception of casts (ie, RBCs, WBCs, fat droplets, crystals, epithelial cells, bacteria, yeast, and sperm), were counted in 10 to 15 microscope fields at a magnification of 40×, and mean number per field was calculated. Casts were counted in 10 to 15 microscope fields at a magnification of 10×, and mean number per field was calculated.

Statistical analyses—The χ^2 test of homogeneity was used to compare frequency of UTI between dogs that received long-term glucocorticoid treatment and dogs that did not. For this analysis, data from only the first urine sample collected from dogs treated with glucocorticoids were used. Unpaired *t* tests or χ^2 tests of homogeneity were used to determine whether type of glucocorticoid, dosage, frequency of administration, or duration of glucocorticoid treatment was associated with UTI. Fisher linear discriminant analysis was used to determine whether results of urinalysis were accurate predictors of UTI; forward stepwise progression was used to determine which values were accurate predictors of UTI in the linear regression model. For development of the linear regression model, dogs treated with glucocorticoids were randomly assigned to 1 of 2 groups. Discriminant analysis was initially performed on one of the groups; the other group was used to evaluate accuracy of the model. The χ^2 test of homogeneity was used to determine whether the number of urine samples submitted for bacterial culture from each dog was associated with detection of UTI and whether sex was associated with detection of UTI. All analyses were performed with standard software[b]; values of $P \leq 0.05$ were considered significant.

Results

A total of 127 dogs with pruritic disorders that had received long-term glucocorticoid treatment were included in the study. Of these, 79 were female (77 spayed and 2 sexually intact) and 48 were male (43 castrated and 5 sexually intact). Age at the time glucocorticoid treatment had been initiated ranged from 11 months to 15 years (mean, 4.7 years). Thirteen dogs were of mixed breeding, and 114 were purebred. Breeds represented included Golden Retriever (n = 21), Labrador Retriever (17), Cocker Spaniel (11), German Shepherd Dog (10), Bichon Frise (8), West Highland White Terrier (6), Fox Terrier (5), Miniature Poodle (3), Miniature Schnauzer (3), Boxer (2), English Bulldog (2), English Springer Spaniel (2), Yorkshire Terrier (2), and 22 other breeds represented by 1 dog each. Pruritic disorders included atopic dermatitis (n = 109 [85.8%]), atopic dermatitis and food allergy (2 [1.6%]), and pruritic disorders of unknown cause (16 [12.6%]). Eighty-nine dogs (70.1%) had been treated with prednisolone, and 38 (30%) had been treated with methylprednisolone. Duration of treatment at the time the first urine sample was collected for bacterial culture ranged from 6.7 months to 7 years (mean ± SD, 1.6 ± 1.1 years). Glucocorticoid dosage ranged from 0.12 to 1.0 mg/kg (0.05 to 0.45 mg/lb), PO, every 48 hours (mean ± SD, 0.28 ± 0.14 mg/kg [0.13 ± 0.06 mg/lb], PO, q 48 h). Only 2 dogs were receiving glucocorticoids at the highest dosage (1 mg/kg, PO, q 48 h). Only 4 dogs were receiving glucocorticoids daily; dosages in these 4 dogs were 0.14 mg/kg (0.06 mg/lb), 0.20 mg/kg (0.09 mg/lb), 0.23 mg/kg (0.1 mg/lb), and 0.24 mg/kg (0.11 mg/lb), PO, every 24 hours.

For 57 of the 127 (45%) dogs, a single urine sample was submitted for bacterial culture during the study period, whereas 2 urine samples were submitted from 46 dogs, 3 urine samples were submitted from 11 dogs, 4 urine samples were submitted from 8 dogs, 5 urine samples were submitted from 4 dogs, and 6 urine samples were submitted from 1 dog. Thus, results of bacterial culture of 240 urine samples were available. The interval between submission of urine samples ranged from 6 to 44 months, with urine samples submitted every 12 to 21 months for 89 (70%) dogs. One hundred eighty-one of the 240 (75.4%) urine samples were collected when the dogs were not receiving antimicrobials for treatment of bacterial skin infections, and 59 (24.6%) samples were collected when dogs were receiving antimicrobials. Fifty-two of the 59 (88%) urine samples collected when dogs were receiving antimicrobials were obtained when dogs were receiving cephalexin (22 mg/kg [10 mg/lb], PO, q 12 h), 4 (7%) were collected when dogs were receiving enrofloxacin (5 mg/kg [2.3 mg/lb], PO, q 24 h), 2 (3%) were collected when dogs were receiving amoxicillin-clavulanic acid (20 mg/kg [9 mg/lb], PO, q 12 h), and 1 (2%) was collected when the dog was receiving sulfadimethoxine-ormetoprim (27.5 mg/kg [12.5 mg/lb], PO, q 24 h). Six urine samples collected when dogs were receiving antimicrobials yielded bacterial growth. All of these urine samples were obtained while dogs were being treated with cephalexin, and all bacterial isolates were resistant to cephalexin in vitro.

None of the owners of the 127 dogs reported that their dogs had clinical signs of UTI at the time samples were collected. However, for 23 of the 127 (18.1%) dogs,

results of bacterial culture of urine samples were positive at least once during the study period (**Table 1**). Multiple urine samples had been collected from 18 of these 23 (78%) dogs, and for 6 of the 18, results of bacterial culture were positive twice. Thus, a total of 29 of the 240 (12.1%) urine samples collected during the study period yielded bacterial growth. For 10 of the 18 dogs from which multiple urine samples were submitted, results of bacterial culture were positive for the first urine sample submitted. For 5 of the 29 (17%) urine samples from which bacterial growth was obtained, 2 species of bacteria were isolated, so that a total of 34 bacterial isolates were obtained. Bacterial growth was obtained for 18 of the 70 (26%) dogs from which > 1 urine sample was collected and for 5 of the 57 (9%) dogs from which only a single urine sample was collected. These proportions were significantly ($P = 0.01$) different.

The 34 bacterial isolates comprised 10 bacterial species. Nonhemolytic *Escherichia coli* was the most common isolate (n = 14), followed by *Enterococcus* spp (6). Susceptibility to 7 antimicrobials was tested, and 31 of 32 (97%) tested isolates were susceptible to trimethoprim-sulfonamide, 25 of 33 (76%) were susceptible to amoxicillin-clavulanic acid, 23 of 33 (70%) were susceptible to tetracycline, 20 of 33 (61%) were susceptible to ampicillin, 17 of 33 (52%) were susceptible to cephalexin, 8 of 31 (26%) were susceptible to chloramphenicol, and 7 of 32 (22%) were susceptible to enrofloxacin.

The proportion of female dogs for which results of bacterial culture were positive (20/79 [25%]) was significantly ($P < 0.007$) higher than the proportion of male dogs for which results of bacterial culture were positive (3/48 [6%]). Urine samples from 17 of the 89 (19%) dogs receiving prednisolone and 6 of the 38 (16%) dogs receiving methylprednisolone yielded bacterial growth; these proportions were not significantly different ($P = 0.7$). Mean ± SD glucocorticoid dosage for the 104 dogs for which results of bacterial culture were negative (0.28 ± 0.15 mg/kg [0.13 ± 0.07 mg/lb], PO, q 24 to 48 h) was not significantly ($P = 0.1$) different from mean dosage for the 23 dogs with positive bacterial culture results (0.23 ± 0.06 mg/kg [0.1 ± 0.03 mg/lb], PO, q 24 to 48 h). Mean duration of glucocorticoid treatment for the 104 dogs with negative bacterial culture results (603 ± 424 days) was not significantly ($P = 0.06$) different from mean duration for the 23 dogs with positive results (798 ± 552 days). Results of bacterial culture were positive for 22 of the 123 (18%) dogs receiving glucocorticoids on an every-other-day basis and for 1 of the 4 (25%) dogs receiving glucocorticoids daily; these proportions were not significantly ($P = 0.7$) different.

When linear discriminant analysis was performed, the presence of pyuria (> 3 WBCs/40X field) was found to correctly predict bacterial growth for 89.9% of the urine samples and the presence of bacteriuria was found to correctly predict bacterial growth for 95.8% of the urine samples. When these parameters were combined, no further improvement in prediction ability was obtained (ie, 95.8% of the urine samples were correctly classified as to bacterial growth). Bacteriuria was not

Table 1—Results of bacterial culture of urine samples from 23 dogs with pruritic disorders that were receiving long-term glucocorticoid treatment.

Dog No.	No. of urine samples	Urine samples that yielded growth	Bacterial species isolated
1	5	First	β-hemolytic *Escherichia coli*
		Fifth	Nonhemolytic *E coli*
2	5	Second	*Klebsiella oxytoca*
3	5	Fourth	Nonhemolytic *E coli*
		Fifth	Nonhemolytic *E coli*
4	4	First	*Staphylococcus intermedius*
5	3	Third	Nonhemolytic *E coli*
6	3	Second	*Enterococcus* spp
		Third	*Enterococcus* spp
7	2	Second	*Klebsiella pneumoniae*
8	2	First	β-hemolytic *E coli*
9	2	First	*Proteus mirabilis*
10	2	Second	Nonhemolytic *E coli*
11	2	First	β-hemolytic *E coli*
		Second	Nonhemolytic *E coli*
12	2	Second	Nonhemolytic *E coli* *Clostridium perfringens*
13	2	First	*Streptococcus* spp
14	2	First	Nonhemolytic *E coli* *Enterococcus* spp
		Second	Nonhemolytic *E coli* β-hemolytic *Streptococcus* spp
15	2	First	*Pseudomonas aeruginosa*
		Second	*Pseudomonas aeruginosa*
16	2	Second	Nonhemolytic *E coli*
17	2	First	*Proteus mirabilis*
18	2	First	Nonhemolytic *E coli* *Enterococcus* spp
19	1	First	Nonhemolytic *E coli*
20	1	First	*Enterococcus* spp
21	1	First	Nonhemolytic *E coli* Group D *Streptococcus* spp
22	1	First	Nonhemolytic *E coli*
23	1	First	*Enterococcus* spp

identified in 7 of the 29 (24%) urine samples that yielded bacterial growth, and pyuria was not identified in 14 of the 29 (48%) samples that yielded growth. Three (10%) samples negative for bacteriuria and pyuria yielded bacterial growth. Interestingly, 3 samples positive for bacteriuria yielded no bacterial growth.

Ninety-four dogs with pruritic disorders that had not received glucocorticoid treatment were included in the study. Of these, 42 were female (40 spayed and 2 sexually intact) and 52 were male (46 castrated and 6 sexually intact). Age at the time of examination ranged from 1 to 14 years (mean, 5.5 years). Sixteen dogs were of mixed breeding, and 78 were purebred. Breeds represented included Labrador Retriever (n = 16), Golden Retriever (15), English Springer Spaniel (4), Staffordshire Bull Terrier (2), Boxer (2), Cocker Spaniel (2), English Bulldog (2), French Bulldog (2), German Shepherd Dog (2), German Shorthair Pointer (2), Great Dane (2), Jack Russell Terrier (2), Pug (2) Standard Poodle (2), and 21 other breeds represented by 1 dog each. Pruritic disorders included atopic dermatitis (65/94 [69%]), atopic dermatitis and food allergy (4 [4%]), food allergy (2 [2%]), and pruritic disorders of unknown cause (23 [24%]). Nine dogs (10%) were receiving antimicrobials at the time urine samples were collected for bacterial culture. Six dogs were receiving cephalexin (22 mg/kg, PO q 12 h), and 1 dog each was receiving enrofloxacin (5 mg/kg [2.3 mg/lb], PO q 24 h), amoxicillin-clavulanic acid (20 mg/kg, PO, q 12 h), and chloramphenicol (45 mg/kg [20.5 mg/lb], PO, q 8 h). None of the owners reported that their dogs had any clinical signs of UTI at the time urine samples were collected, and none of the urine samples yielded bacterial growth.

For comparisons of the 2 groups, only data from the first urine sample collected from dogs treated with glucocorticoid were used. The proportion of dogs treated with glucocorticoids with positive bacterial culture results (15/127 [12%]) was significantly ($P < 0.005$) higher than the proportion of dogs that had not received glucocorticoids (0/94 [0%]).

Because of differences in sex distribution between groups of dogs that had and had not been treated with glucocorticoids, we compared females and males separately. Females treated with glucocorticoids were significantly ($P < 0.005$) more likely to have positive bacterial culture results than were females that had not received glucocorticoids. However, males treated with glucocorticoids were not significantly ($P = 0.07$) more likely to have positive bacterial culture results than were males that had not received glucocorticoids.

Discussion

In the present study, 23 of 127 (18.1%) dogs with pruritic disorders that had received long-term glucocorticoid treatment developed a UTI, whereas none of the 94 dogs with similar disorders that had not been treated with glucocorticoids had a UTI. This suggests that long-term glucocorticoid treatment may predispose dogs to develop UTIs.

A previous study[3] reported a much higher frequency of UTI (39%) in dogs receiving long-term glucocorticoid treatment to control various skin disorders than was found in the present study. The higher rate of infection in this previous study might be explained by differences in the population of dogs and in the dosages of glucocorticoids that were administered. Thirty percent of the dogs in the previous study had autoimmune skin disease, which usually requires higher dosages of glucocorticoids to maintain adequate disease control. In fact, the mean glucocorticoid dosage for dogs receiving every-other-day treatment in this previous study was 0.8 mg/kg (0.36 mg/lb), compared with 0.28 mg/kg in the present study. Additionally, 35% of the dogs in the previous study received glucocorticoids on a daily basis, compared with only 3% of the dogs in the present study. In the present study, 24% of the urine samples were collected when dogs were receiving antimicrobials, whereas none of the dogs were receiving antimicrobials in the previous study. The frequency of UTI in the present study would have been 21% if dogs that were receiving antimicrobials had been excluded. However, we believe that these dogs should be included because dogs with pruritic disorders that receive long-term glucocorticoid treatment often are treated with antimicrobials to manage recurrent skin bacterial infections. Further, our findings indicate that the possibility of UTI should not be dismissed in dogs receiving glucocorticoids simply because they are also being treated with antimicrobials. Our results corroborate the finding of the previous study[3] in that type of glucocorticoid, dosage, and duration and frequency of drug administration were not significantly associated with frequency of UTI. However, the *P* value used to test for an association between duration of treatment and frequency of UTI was close to the cutoff for significance (0.06), and it is possible that if more dogs had been included in the study, a significant difference would have been demonstrated. As was the case in previous studies,[3,11-13] the frequency of UTI was significantly higher in females than in males.

In the present study, results of discriminant analyses suggested that bacteriuria and pyuria were accurate predictors of UTI. However, bacteriuria was not identified in 24% of urine samples that yielded bacterial growth. Therefore, urinalysis alone is not sufficient to screen dogs receiving long-term glucocorticoid treatment for UTI. Moreover, 3 samples without bacteriuria or pyuria yielded bacterial growth, reinforcing the need for bacterial culture of urine samples from dogs receiving long-term glucocorticoid treatment even when results of a urinalysis do not indicate the presence of infection. Interestingly, 3 samples with bacteriuria yielded no growth. This can be explained by contamination of the urine sample after collection, an incorrect laboratory interpretation, inadequate culture technique, or the presence of nonviable bacteria in the urine.[14] One of these dogs was receiving amoxicillin-clavulanic acid at the time the urine sample was collected, which could have caused bacteria in the urine to be nonviable.

None of the owners of the dogs included in the present study reported seeing any clinical signs of UTI; however, owners of dogs treated with glucocorticoids were not routinely asked specific questions related to clinical signs of UTI. It is possible that some of the dogs had mild clinical signs of UTI, which the owners failed to recognize or chose not to report. Nevertheless, our results and previous data[3,8] show that depending on

owners to report clinical signs of urinary tract disease will lead to underdiagnosis of UTI in these patients.

Urinalyses and bacterial culture of urine samples were performed as part of a systematic approach adopted by the University of Minnesota Dermatology Service to monitor dogs for potential adverse effects associated with long-term glucocorticoid treatment. Therefore, we believe that the population of dogs included in this study was unbiased with regard to the chance that they would develop UTI. Moreover, the absence of UTI among dogs with pruritic disorders not receiving glucocorticoids supports the view that dogs that receive long-term glucocorticoid treatment are at increased risk of developing UTI. However, this comparison must be made with caution because urine samples were not collected concurrently from the 2 groups, and although unlikely, it is possible that a higher frequency of UTI occurred during the time urine samples were collected from dogs receiving glucocorticoids.

The larger number of females in the glucocorticoid-treated group could be argued as the reason for the significantly higher frequency of UTI in this group, compared with dogs that were not treated with glucocorticoids. However, when females were analyzed separately, the difference between groups was still significant, suggesting that glucocorticoid treatment, not sex, was the predisposing factor for the development of UTI. When males were analyzed separately, no difference in frequency of UTI was found between dogs that were or were not treated with glucocorticoids. However, because males were less predisposed to UTI than females, only 3 glucocorticoid-treated male dogs had UTI, limiting our ability to identify a significant difference.

At least 2 urine samples were submitted for bacterial culture from 70 of the 127 (55%) dogs receiving long-term glucocorticoid treatment in the present study, and this increased the chances of detecting UTI. Thus, our results suggest that bacterial culture of serial urine samples was more likely to detect UTI than was culture of a single urine sample. Therefore, we recommend that urine samples be submitted for bacterial culture at least once a year when dogs are receiving glucocorticoid treatment.

Nonhemolytic *E coli* was the most common isolate in the present study, accounting for 14 of the 34 (41%) bacterial isolates obtained. This is consistent with results of previous studies[3,8,15-18] in which *E coli* was the most common bacterial pathogen of the canine urinary tract. The percentage of urine samples yielding > 1 species of bacteria was similar to percentages in previous reports.[3,8]

Only 22% of the isolates were susceptible to enrofloxacin in the present study. This low frequency may reflect the common use of this antimicrobial in veterinary medicine. Additionally, only 51% of the isolates were susceptible to cephalexin, an antimicrobial frequently used to manage bacterial skin infections. In contrast, 97% of the isolates were susceptible to trimethoprim-sulfonamide and 76% were susceptible to amoxicillin-clavulanic acid. These findings likely reflect the infrequent use of these 2 antimicrobials to manage bacterial skin infections in dogs at our institution.

Specific tests to identify underlying concurrent disorders that could have predisposed dogs to development of UTI, such as urolithiasis, urinary tract neoplasia, renal disease, and diabetes mellitus, were not performed in the present study. However, none of these dogs had clinical signs associated with these diseases. Moreover, dogs that were not treated with glucocorticoids were as likely to have these diseases as were dogs treated with glucocorticoids.

In summary, results of this study confirm that dogs receiving long-term glucocorticoid treatment are at increased risk for UTI even if the drug is administered at a low dosage on an every-other-day schedule. Therefore, we recommend that urine samples be routinely submitted for bacterial culture at least yearly in dogs receiving long-term glucocorticoid treatment.

a. Sensititre, Trek Diagnostic Systems Inc, Westlake, Ohio.
b. SPSS, version 11.5.1, SPSS Inc, Chicago, Ill.

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