Intermittent alkaline urine in a cat fed an acidifying diet

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A 2-year-old spayed female domestic shorthair cat was referred for evaluation of chronic stranguria and urinating outside the litter box (inappropriate urination). The cat was kept indoors with another cat, and was provided a litter box containing generic litter. The client had owned the cat since it was a kitten, and signs of lower urinary tract disease (inappropriate urination and stranguria) were first noticed when the cat was approximately 6 months old. Since the initial episode, there had been 2 subsequent episodes lasting longer than 10 days. The cat had been evaluated and treated by 3 other veterinarians with limited success. Treatments had included administration of enrofloxacin, amoxicillin trihydrate/clavulinate potassium, and unidentifiable corticosteroids. The cat had been fed a calcolycytic dry diet for approximately 1 year after the first appearance of signs of lower urinary tract disease, at which time the diet was changed to a diet designed to correct the obesity the cat had developed while it was being fed the calcolycytic diet.

On physical examination, the cat weighed 6.2 kg, had a rectal temperature of 39.5°C, and was tachypneic (80 breaths/min). The high respiratory rate and rectal temperature were attributed to the cat’s apparent anxiety. Laboratory evaluation included CBC, serum biochemical analysis, determination of venous blood gases, urinalysis, and bacteriologic culture of urine obtained by cystocentesis. Plain abdominal radiography, double-contrast cystography, and urethrography were performed. Abnormal serum biochemical test results included hypokalemia (3.3 mEq/L), mild hyperchloremia (126 mEq/L; 153 mEq/L sodium), low serum bicarbonate concentration (16.5 mEq/L); venous pH, 7.34; P<sub>CO</sub>₂, 31.4 mm of Hg; and high values for serum creatinine (2.2 mg/dl) and creatinine kinase activity (1,676 IU/L). Bacteriologic culturing did not yield growth, and abnormalities were not detected by double-contrast cystography-urethrography. Urine pH was 6.0 by dipstick, 6.95 by pH meter. Urine specific gravity was 1.026, and a trace of protein was found by dipstick evaluation. Microscopic evaluation of urine sediment revealed 0 to 1 leukocytes, 0 to 1 erythrocytes, and 0 to 2 epithelial cells/field at 400X magnification.

A diagnosis of idiopathic lower urinary tract disease was made, and a different dry diet (designed to prevent struvite crystal formation) was prescribed. Amoxicillin (150 mg, PO, q 12 h) was dispensed in an effort to prevent iatrogenic bacterial urinary tract infection after the instrumentation necessary to perform urethroctography. The origin of the high creatine kinase activity could not be determined, but may have resulted from an IM injection of ketamine given prior to blood sampling.

When the cat was reevaluated 2 weeks after the initial evaluation, the owner reported that there was much improvement in the cat’s condition. Although the cat had urinated outside the litter box twice, there was no stranguria. The only abnormality observed on physical examination was tachypnea (72 breaths/min). Laboratory evaluation consisted of analyses of urine, obtained by cystocentesis, and venous blood gases. All results were within reference ranges for our laboratory, including serum bicarbonate. Urine pH was 6.0 by dipstick, 6.56 by pH meter.

Four months after initial evaluation, the cat was reexamined because it had had intermittent hematuria, stranguria, and inappropriate urination of 1 week’s duration. At this time, the cat weighed 7.3 kg, but there were no abnormal physical findings. The cat was more aggressive than it had been during previous examinations. Laboratory abnormalities included increased serum creatine kinase activity (1,756 IU/L), 3+ microhematuria, and struvite crystaluria. Urine pH was 8.0 by dipstick, 7.91 by pH meter.

The finding of alkaline urine was unexpected. Causes of alkaline urine include bacterial urinary tract infection, vomiting, ingestion of an alkalinizing diet, metabolic alkalosis, drug ingestion, cystal renal tubular acidosis, and hyperventilation. Bacterial urinary tract infection was ruled out on the basis of inability to isolate bacteria from the urine. Vomiting, ingestion of an alkalinizing diet, drug ingestion, and metabolic alkalosis were ruled out on the basis of interview of the clients, the fact that the cat had been deprived of food for longer than 12 hours prior to examination, and venous blood gas results (pH, 7.30; HCO<sub>₃</sub>-, 17.8 mEq/L; P<sub>CO</sub>₂, 37.2 mm of Hg). Distal renal tubular acidosis was considered unlikely on the basis of lack of signs of lethargy, depression, anappetence, metabolic acidosis, and signs referable to renal disease described in case reports of cats with this disease. Hyperventilation, possibly induced by stress, was considered the most likely explanation for the alkaline urine, based on the low probability of other recognized

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causes, and on the dysphoria and tachypnea observed by the owners during transport of the cat as well as its agitation observed by us.

To test this hypothesis, the owners were provided with nonabsorbable litter and a urine collection tube containing thymol-saturated mineral oil to collect a urine specimen the day before returning the cat for reexamination in 1 month. On subsequent examination of the cat, the owners again reported that the cat had appeared distressed during transit. Because of the aggressiveness of the cat, rectal temperature and pulse and respiratory rates were not determined, blood gases were not collected, and urine was collected through a cystoscope after the cat was anesthetized for cystoscopic evaluation of the urethra and urinary bladder.

Laboratory evaluation consisted of urinalysis and bacterial culture of urine obtained by cystocentesis; all results were found to be within reference ranges. Urine pH of the sample collected by the owners the day before admission was 5.5 by dipstick and 6.19 by pH meter, whereas the pH of urine collected at cystoscopy after 14 hours of food deprivation was 7.5 by dipstick, 7.65 by pH meter. At cystoscopy, no tumors, uroliths, or anatomic abnormalities were observed, whereas submucosal petechial hemorrhages and edema consistent with interstitial cystitis were observed.

Despite multiple signs of lower urinary tract disease, alkaline urine, and struvite crystalluria, this cat did not have struvite urolithiasis. The lack of association between crystalluria and struvite urolithiasis supports the findings reported by Kruger et al., that the frequency of crystalluria in the urine of nonobstructed cats with lower urinary tract disease was not different from that of clinically normal control cats. The lack of agreement between dipstick and pH meter determinations of urine pH we observed also has been reported by others.7

More surprising is the finding that this cat produced sterile, alkaline urine and struvite crystals, in the absence of an identifiable cause, despite consuming a urine-acidifying diet. This result led us to compare the pH of urine collected at home with that collected at the clinic. There was a 1.4-unit difference in pH (a 25-fold decrease in urine hydrogen ion activity). On the basis of the cat's dysphoria and "panting" during transport to the clinic, as reported by the owners, the most likely cause of the increase in urine pH was anxiety-induced hyperventilation.8 Hyperventilation in dogs8 results in sustained increases in urine pH of approximately 1 pH unit. Additionally, IV infusions of adrenaline and noradrenaline have resulted in increases in respiratory minute volume of 14 and 8%, respectively, in anesthetized cats.9 This increment in respiratory minute volume is comparable to that reported in human beings with hyperventilation syndrome. Moreover, voluntary hyperventilation in human beings has been reported to increase urine pH by more than 1 pH unit within 3 hours.10

On the basis of our experience with the cat of this report, we conclude that alkaline urine may be encountered in cats fed urine-acidifying diets. Some cats may produce alkaline urine during periods of acute stress, possibly secondary to hyperventilation. Unfortunately, acute respiratory alkaloisis could not be proven in the cat of this report because of our inability to collect the necessary samples until the cat was anesthetized. Low Pco₂ is a tubular signal to absorb less bicarbonate from tubular fluid and could account for an alkaline urine pH.11 Other mechanisms related to stress also could be involved, such as an influence of the sympathetic nervous system on renal tubular acid-base dynamics. For example, Schlegel12 reported that IV administration of adrenaline or noradrenaline to rabbits caused an increase in urine pH of more than 1 unit that continued after administration of the catecholamines ceased. Because of possible confounding effects of stress on the evaluation of urine pH in cats, we recommend that veterinarians finding high urine pH in cats fed acidifying diets that do not have urinary tract infections consider repeating their evaluation with urine collected by the owners at home.

References