Serum biochemical abnormalities in goats with uroliths: 107 cases (1992–2003)

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Objective—To characterize serum biochemical abnormalities in goats with uroliths.

Design—Retrospective case-control series.

Animals—107 male goats with uroliths and 94 male goats with various nonrenal diseases (controls).

Procedures—For male goats, results of serum biochemical analyses collected from 1992 through 2003 were retrieved from computerized records, as were signalment, clinical diagnoses, and discharge status. Results of analyses for BUN, creatinine, phosphorus, calcium, Na, K, Cl, total CO_2, anion gap, and glucose were compared between goats with uroliths and control goats.

Results—Goats with uroliths had higher mean BUN, creatinine, total CO_2, K, and glucose concentrations and lower mean phosphorus, Na, and Cl concentrations than control goats, with no difference in mean calcium concentration and anion gap. Goats with uroliths had higher frequency of azotemia, hypophosphatemia, hypochloridemia, and increased total CO_2 and lower frequency of decreased total CO_2 than control goats. Urolithiasis occurred more frequently in castrated males than in sexually intact males and in dwarf African breeds than in other breeds.

Conclusions and Clinical Relevance—Goats with uroliths often had hypophosphatemia at admission. Hypochloridemic metabolic alkalosis was the most common acid-base disorder. Rupture in the urinary tract system was associated with increased prevalence of hyponatremia and hyperkalemia. Clinicians should be aware of these abnormalities when determining fluid therapy. (*J Am Vet Med Assoc* 2007;230:101–106)

RUMINANTS/ CAMELIDS

Obstructive urolithiasis is a common condition of male goats that is inevitably fatal without medical or surgical intervention[1-5] Affected animals may have urethral obstruction alone or develop secondary complications of ruptured bladder and uroperitoneum or ruptured urethra with subcutaneous urine pooling.[5] Laboratory abnormalities associated with obstructive urolithiasis in most nonruminant species include azotemia, hyperphosphatemia, hyponatremia, hyperkalemia, and metabolic acidosis.[6-9] Urolithic cattle have similar abnormalities, except that potassium and phosphorus may be either increased or within reference intervals; cattle also may have metabolic alkalosis.[10-13] Azotemia is the only well-documented serum biochemical abnormality in urolithic goats.[3,4,14,15] Because treatment often includes surgery with general anesthesia, knowledge of expected serum biochemical abnormalities is important in patient care. The purpose of the study reported here was to characterize the serum biochemical abnormalities in urolithic goats to determine whether they are similar to those reported in other species.

Criteria for Selection of Cases

Goats were included in the study if they had urolithiasis and if blood had been collected for serum biochemical analysis on entry to the hospital.

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Abbreviations

UCDVMTH	University of California-Davis Veterinary Medical Teaching Hospital
T_{CO_2}	Total CO_2
GFR	Glomerular filtration rate

Procedures

Electronic medical records from January 1992 through December 2003 of the UCDVMTH were searched for the first serum biochemical panel performed at the initial visit of male goats > 6 months of age. Other information collected from each patient record included signalment, discharge diagnosis, and clinical outcome (alive, died, or euthanized). Diagnoses were categorized as urolithiasis (n = 107) or nonrenal disease (94). The few goats (n = 6) with primary renal disease were eliminated from statistical analysis because

Table 1—Sex and breed distributions (No. [%]) of male goats with urolithiasis or nonrenal diseases.

Variable	Urolithiasis	Nonrenal diseases
Sex (n = 201 goats)		
Sexually intact male	21 (19.6)	43 (45.7)
Castrated male	86 (80.4)	51(54.2)
Breed type (185)		
Dairy meat	44 (44.4)	54 (62.85)
Dwarf African	50 (50.6)	28 (32.6)
Rare breeds	5 (5.0)	4 (4.6)

Table 2—Mean ± SD serum biochemical values in 107 goats with urolithiasis and 94 goats with various nonrenal conditions.

Variable	Urolithiasis	Nonrenal diseases	*P* value	Reference range
BUN (mg/dL)	73.2 ± 68	26 ± 68	< 0.001	19–31
Creatinine (mg/dL)	6.3 ± 6.8	1.3 ± 0.9	< 0.001	0.7–1
Calcium (mg/dL)	9.1 ± 1.0	8.9 ± 1.2	NS	9.2–11.7
Phosphorus (mg/dL)	4.0 ± 3.2	6.2 ± 4.8	0.002	4.2–9.1
Na (mmol/L)	144.6 ± 5.3	146.7 ± 5.5	< 0.001	140–150
K (mmol/L)	4.5 ± 1.2	4.2 ± 0.7	0.006	3.4–5.7
Cl (mmol/L)	101.6 ± 9.2	106.9 ± 7.5	< 0.001	101–112
TCO_2 (mmol/L)	27.8 ± 6.1	23.9 ± 6.1	< 0.001	22–28
Anion gap (mmol/L)	19.8 ± 7.8	20.0 ± 8.7	NS	11–25
Glucose (mg/dL) *	149.3 ± 62.8	117.3 ± 55.6	0.003	52–81

*n = 93 for urolithiasis group and 92 for nonrenal diseases group. NS = Not significant ($P \geq 0.05$).

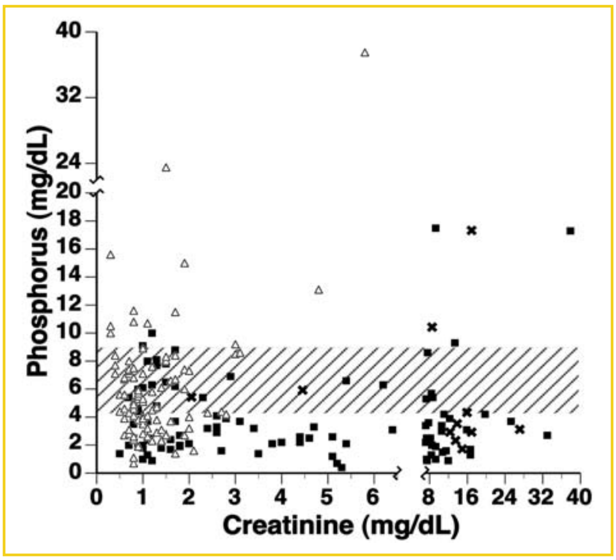

Figure 1—Relationship between serum phosphorus and creatinine concentrations in male goats with nonrenal diseases (triangles), urolithiasis (squares), and urolithiasis with urinary tract rupture (X). Creatinine values to the right of the break in the x-axis were detected in urolithic goats only. Phosphorus values above the break in the y-axis were detected in goats with nonrenal diseases only. The rectangle formed by diagonal lines represents the reference range for phosphorus concentration.

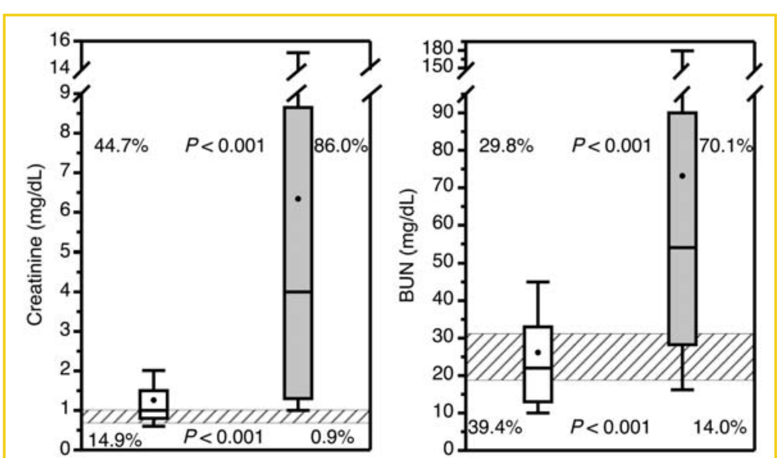

Figure 2—Box-and-whisker plots of serum concentrations of creatinine and BUN in goats with urolithiasis (shaded box) or nonrenal diseases (white box). Whiskers indicate 90th and 10th percentiles for each group, boxes indicate 75th to 25th percentiles, horizontal bars indicate population medians, and dots indicate mean values. Diagonal lines indicate reference ranges. Percentage values indicate proportion of a population with values greater or less than the reference range. *P* values indicate comparison of frequency of abnormal results (ie, greater or less than reference limits) between the 2 groups.

of their low number. On the basis of medical record information, goats were categorized according to breed type as dairy-meat (eg, Nubian, Alpine, Toggenburg, and Boer), dwarf African (Pygmy and Dwarf Nigerian), various rare breeds (eg, Angora and San Clemente), and unknown.

Serum analytes evaluated included BUN, creatinine, total calcium, inorganic phosphorus, Na, K, Cl, TCO_2, and glucose. Anion gap was calculated according to a formula ($Na + K - Cl - TCO_2$). Analytes of the urolithic and nonrenal disease goats were compared via differences in mean concentrations and the frequency of results outside of reference ranges for healthy goats provided by the UCDVMTH Clinical Chemistry Laboratory.

Statistical analysis—The unpaired *t* test was used for comparison of means, and the χ^2 test or Fisher exact test was used for comparisons of categoric data. Frequencies of abnormalities among urolithic goats with uncomplicated urethral blockage, ruptured bladder, or ruptured urethra were compared via stepwise logistical regression. Values of $P < 0.05$ were considered significant.

Results

Population characteristics—Overall, 68.2% of goats in this study had been castrated. Castrated goats comprised a significantly ($P < 0.001$) greater proportion of urolithic goats (80.4%) than in the population with nonrenal diseases (54.2%). Breed information was available for 185 of the 201 goats. Dwarf African goats were more likely to be evaluated for urolithiasis than either dairy-meat–type breeds or rare breeds ($P = 0.04$). There was no interaction between castration status and the likelihood of a breed type to develop urolithiasis ($P = 0.3$; **Table 1**). Ninety-six urolithic goats were discharged alive, whereas 11 did not survive (10 were euthanized and 1 died). Survival rate for urolithic goats was significantly ($P = 0.003$) higher than that of the nonrenal diseases population, which had 69 survivors and 25 nonsurvivors.

Ninety-five of the urolithic goats had urethral obstruction alone, whereas 7 also had a ruptured bladder and 5 had a ruptured urethra. Goats with secondary urinary tract rupture were significantly ($P = 0.02$) more likely to have a fatal outcome

(4 dead/12 goats), compared with uncomplicated obstruction (7 dead/95 goats).

Serum biochemical analysis—The most striking differences between urolithic goats and goats with nonrenal diseases were the significantly higher mean BUN and creatinine concentrations and lower phosphorus concentrations in urolithic goats (**Table 2**). Phosphorus concentration was low in many urolithic goats over the whole range of creatinine values, whereas it was often increased along with creatinine in the comparison population (**Figure 1**). There was no significant difference in mean calcium values, although the means for both populations were slightly below the reference interval. Urolithic goats had slightly lower mean Na and Cl concentrations and higher K and TCO_2 concentrations than control goats, with no difference in mean anion gap. Both populations had mean glucose concentrations much greater than the reference interval, and urolithic goats had significantly higher glucose than goats with nonrenal diseases.

Comparisons of the frequencies of abnormal laboratory results were made (**Figures 2–6**). Urolithic goats had a higher frequency of hypercreatinemia and high BUN and a lower frequency of hypocreatinemia and low BUN, compared with nonrenal disease goats. Most of the urolithic goats were hypophosphatemic (67%), compared with 38% of nonrenal disease goats. Hyperphosphatemia was uncommon and similar in both populations. Hypocalcemia occurred in most goats without significant differences in prevalence between urolithic goats and the nonrenal disease goats. Hypercalcemia was extremely rare (n = 3 goats), and no statistical comparison was done for that abnormality.

Urolithic goats had a higher frequency of hyperkalemia, hypochloridemia, and high TCO_2 and a lower frequency of low TCO_2, compared with nonrenal

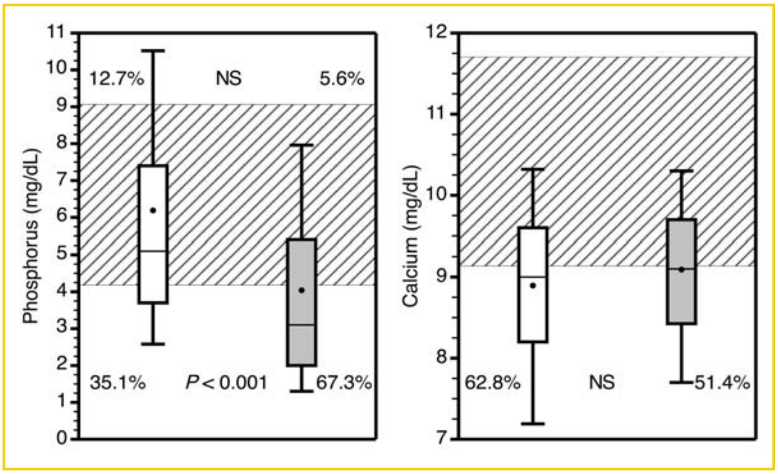

Figure 3—Box-and-whisker plots of serum concentrations of phosphorus and calcium in goats. *See* Figure 2 for key. NS = Not significant ($P \geq 0.05$).

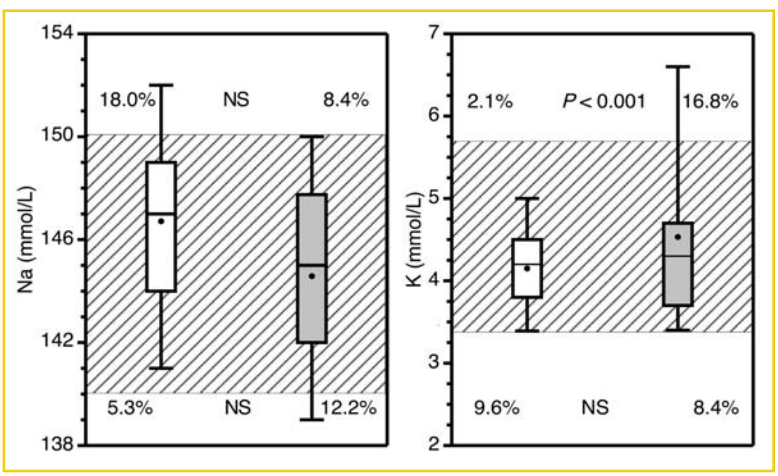

Figure 4—Box-and-whisker plots of serum concentrations of sodium and potassium in goats. *See* Figures 2 and 3 for key.

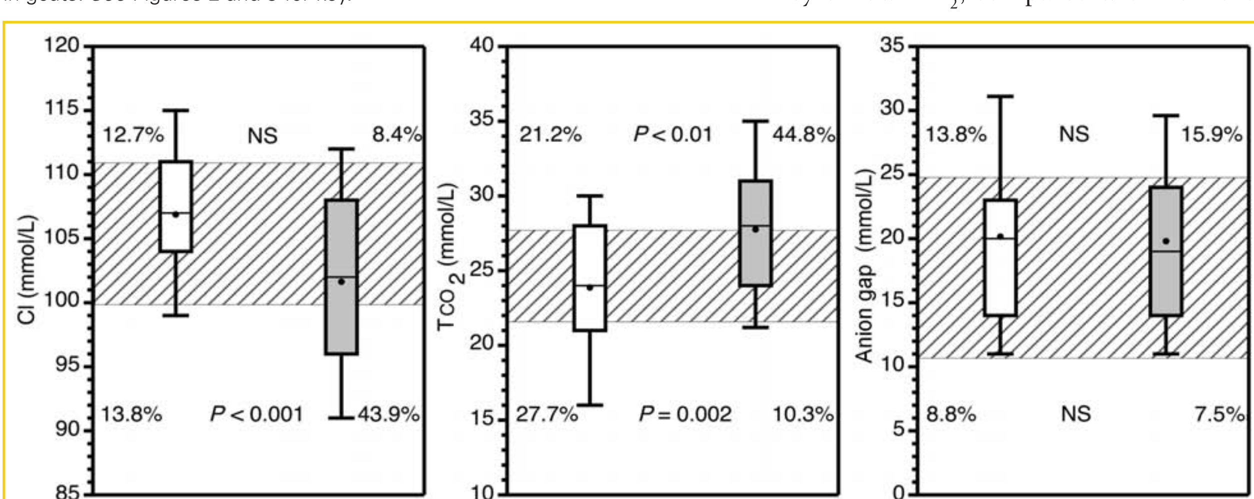

Figure 5—Box-and-whisker plots of serum concentrations of chloride, TCO_2, and anion gap in goats. *See* Figures 2 and 3 for key.

disease goats. For Na and anion gap, the frequencies of abnormalities were low and there were no significant differences between the populations. Hyperglycemia was highly prevalent, with equal frequency in both populations, whereas urolithic goats were less likely to be hypoglycemic.

In the urolithic goat population, secondary urinary tract rupture resulted in significant differences in the frequency of abnormalities, compared with goats with uncomplicated urethral obstruction (**Table 3**). Goats with any type of urinary tract rupture were more likely to have azotemia, hyperkalemia, and hypochloridemia than were goats with uncomplicated urethral obstruction. In addition, goats with ruptured bladder were more likely to have hyponatremia and hypophosphatemia, abnormal T_{CO_2}, and increased anion gap, compared with goats with urethral blockage alone.

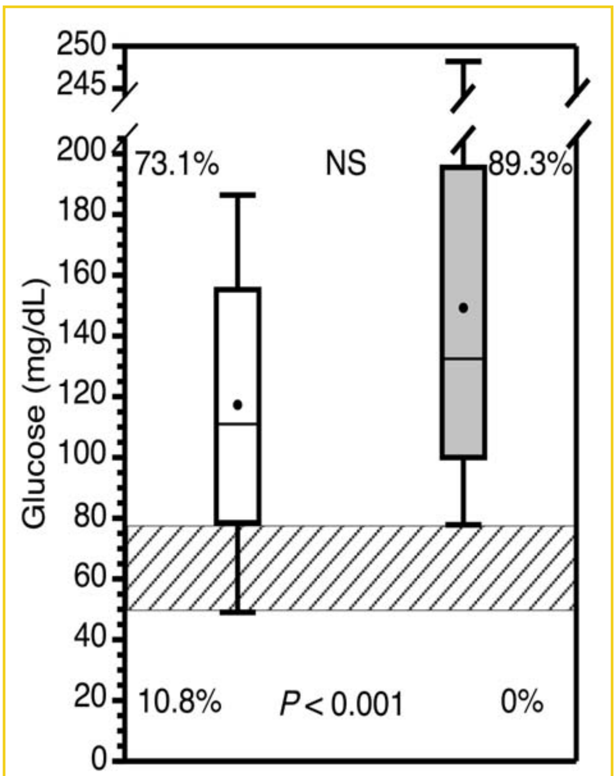

Figure 6—Box-and-whisker plots of serum concentrations of glucose in goats. *See* Figures 2 and 3 for key.

Discussion

The high prevalence of hypophosphatemia in urolithic goats was an unexpected finding because hyperphosphatemia is the expected result of postrenal obstruction or ruptured bladder in most domestic species.[6,7,9-13,16,17] Only horses are similar to goats, with hypophosphatemia being characteristic of postrenal obstruction.[18,19] The high prevalence of hypophosphatemia in these urolithic goats (67.3%) appeared to be associated with the condition because it was significantly higher than the prevalence in male goats that were ill from other conditions (37%). The hypophosphatemia could not be ascribed to early diagnosis and minimal metabolic derangement because it occurred in many severely azotemic goats, including goats with secondary urinary tract rupture (Figure 1). These results differed from previously published studies that found either no pattern of phosphorus abnormalities in experimental urethral obstruction[20] or hyperphosphatemia in a combined population of goats and sheep with clinical urolithiasis.[21]

Obstructive urolithiasis in most nonruminant species leads to hyperphosphatemia because renal excretion is the primary route for elimination of excess phosphorus.[6-9] In contrast, healthy ruminants excrete little phosphorus through their kidneys because most excess phosphorus is eliminated through the gastrointestinal tract. Ruminant saliva contains a large amount of phosphorus to ensure sufficient supply of this element to the rumen microflora.[22,23] Salivary phosphorus loss is balanced by a high rate of intestinal phosphorus absorption, with excess phosphorus lost in the feces. Endogenous phosphorus recycling is facilitated by the salivary glands' lower threshold for plasma phosphorus uptake, compared with the renal excretory threshold.[24] For many ruminants, almost all phosphorus in the glomerular filtrate is reabsorbed in the proximal tubules.[23] Ingestion of a high-phosphorus diet is 1 cause of increased urinary phosphorus excretion.[25] Decreased saliva production, caused either by ingestion of finely pelleted feeds[26] or ligation of the parotid salivary duct,[27] diverts phosphorus excretion to the kidneys and results in increased phosphaturia.[23]

Although the urinary system is a minor route for phosphorus excretion in healthy ruminants, decreased GFR in cattle and sheep often causes hyperphosphatemia.[28-31] Apparently, in these situations, less phosphorus is lost through the gastrointestinal tract because de-

Table 3—Proportions of 107 urolithic goats that had serum biochemical values that were significantly ($P < 0.05$) different among those without urinary tract rupture (n = 95), those with ruptured bladder (7), and those with urethral rupture (5).

Variable	No urinary tract rupture	Ruptured bladder	Ruptured urethra	*P* value
Increased creatinine	80/95	7/7	5/5	< 0.001
Increased BUN	64/95	6/7	5/5	< 0.001
Decreased phosphorus	61/95	5/7	2/5	< 0.001
Decreased Na	8/95	4/7	1/5	< 0.001
Increased K	12/95	3/7	3/5	< 0.001
Decreased Cl	38/95	6/7	3/5	< 0.001
Increased T_{CO_2}	42/95	4/7	2/5	0.005
Increased anion gap	12/95	4/7	1/5	0.02
Decreased T_{CO_2}	8/95	3/7	0/5	0.001

creased rumination reduces saliva production. Excess phosphorus is diverted to the kidneys for excretion, but it is retained just as it is in most monogastric animals with decreased GFR.[32] In the present study, hyperphosphatemia was detected in many of the azotemic goats of the comparison group, similar to the situation for cattle with prerenal azotemia.[28] For most of the urolithic goats, however, a different metabolic balance for phosphorus must have occurred, preventing hyperphosphatemia in most of them despite decreased GFR.

Although relative unimportance of renal phosphorus excretion might explain the relative infrequency of hyperphosphatemia in urolithic goats, it cannot explain the high prevalence of hypophosphatemia in these goats. One possibility is that they were being fed a low-phosphorus diet, a known cause of total body phosphorus deficiency and hypophosphatemia in goats.[33] Secretion of corticosteroids, epinephrine, or a combination of both from pain and stress could cause the hypophosphatemia found in this population. Administration of corticosteroids to goats induces hypophosphatemia within 12 hours.[34] In humans, epinephrine causes a rapid decrease in serum phosphorus concentration, probably because of shifts into the intracellular space.[35] The hyperglycemia found in most of the urolithic goats might have triggered hyperinsulinemia, which causes hypophosphatemia because of the phosphorus shift into the intracellular space.[36] We could not eliminate the possibility that an interfering substance caused falsely low phosphorus concentration because specific validation of the laboratory's phosphorus assay for goat serum was not performed. This possibility was unlikely; few substances are known to interfere with the molybdinate technique used by the laboratory.[37] Furthermore, because it is unlikely that any of these possible causes of hypophosphatemia would occur more frequently in urolithic goats than in the control population, the reason for the association between urolithiasis and hypophosphatemia remains unexplained.

Some human patients with recurrent urolithiasis from calcium-containing calculi are hypophosphatemic, yet have inappropriately high concentrations of urinary phosphorus.[38] Prie et al[39] propose that this syndrome is caused by a deficiency in renal reabsorption of phosphorus (ie, renal phosphate leakage). The association between hypophosphatemia and urolithiasis found in the present study raises the possibility of a similar renal phosphate leak that caused at least some instances of urolithiasis. Goats of dwarf African breeds with increased risk for urolithiasis are also support for a possible inherited disorder that predisposed to urolith formation. Investigations of phosphorus balance in goats with urolithiasis that include evaluation of fecal and renal fractional excretion are required to determine whether inappropriate excretion of phosphorus contributes to urolithiasis in this species.

The high prevalence of hypochloridemic metabolic alkalosis (decreased Cl and increased T_{CO_2} concentrations) in the goats with uncomplicated urethral blockage was similar to the situation in cattle with renal diseases.[30] Hyponatremia and hyperkalemia and metabolic acidosis (low T_{CO_2}) were relatively infrequent, but were higher in frequency when associated with uroperitoneum. Similar abnormalities have been found in other species with clinical or experimentally induced uroperitoneum.[8,10,12]

Although a previous clinical study[14] found secondary urinary tract rupture to be a common complication of urolithiasis, most of the goats reported here (n = 95) had uncomplicated urethral blockage. It appeared that goat owners in our practice area were aware of the dangers of urethral calculi and sought veterinary care before the development of urinary tract rupture. Early treatment was associated with fewer electrolyte abnormalities and higher survival rate, with 92.4% of goats with uncomplicated urethral obstruction discharged alive.

Results of this study should help clinicians in determining appropriate treatment for urolithic goats. Phosphorus supplementation may be required for goats with hypophosphatemia. Although mild hypophosphatemia may cause no apparent clinical consequences, severe hypophosphatemia may cause general muscle weakness, hemolytic anemia, rhabdomyolysis, or serious cardiac abnormalities.[40] Postsurgical intravascular hemolysis has been seen rarely in urolithic goats at UCDVMTH.[a] The finding that the most frequent electrolyte abnormalities included hypochloridemic metabolic alkalosis and hyperkalemia should help clinicians determine appropriate fluid therapy.

Results of this study offer some insights into laboratory abnormalities in male goats, irrespective of their disease. Hyperglycemia was the most frequent abnormality and was often quite marked. The high prevalence of hypocalcemia was an unexpected finding. Recently, hypocalcemia has been recognized as a frequent abnormality in hospitalized humans, cats, and dogs that are severely ill with numerous diseases.[41-43] It may be that hypocalcemia is a fairly frequent complication of severe illness in goats.

Although this retrospective case-control series identified hypophosphatemia as a common abnormality in caprine urolithiasis, it was limited by lack of information on the phosphorus status of the goats prior to developing clinical urolithiasis. Prospective studies that include serum and urinary phosphorus measurements and determination of dietary phosphorus content are needed to determine the relationship between phosphorus balance and development of uroliths in goats.

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