Ammonium urate urolith resulting in hydronephrosis and hydroureter in a dog with a congenital portosystemic shunt

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A four-year-old, female Yorkshire terrier was referred to the University of Missouri Veterinary Teaching Hospital for evaluation of cranial trauma two weeks after the dog had been struck on the head by a golf ball. The client noted that, after the blow, the dog was depressed, polydipsic, anorectic, and seemed to have intermittent episodes of blindness. Treatment with steroids had resulted in marginal improvement in the dog's attitude. Vaccination status was up-to-date, and there was no known exposure to toxins.

On examination, the rectal temperature, heart rate, and respiratory rate were within normal limits. The dog was depressed, restless, and drooled. The physical and ophthalmoscopic examinations were otherwise unremarkable. Slight ataxia and conscious proprioceptive deficits in both hindlimbs were detected on the neurological examination.

The abnormal behavior, neurological abnormalities, and history of cranial trauma were suggestive of a cerebral disorder. Differential diagnoses included hydrocephalus, trauma, meningoencephalitis, and toxic or metabolic encephalopathy. Skull and thoracic radiographs were within normal limits. There was no evidence of hydrocephalus on an electroencephalogram. A small liver was identified on abdominal radiographs. The results of a complete blood count (CBC), serum biochemical profile, and urinalysis suggested hepatic insufficiency (nonregenerative anemia, decreased plasma proteins, decreased serum urea levels, and ammonium urate crystals in the urine). A resting serum ammonia level was markedly elevated (157 μmol/L). The tentative diagnosis was hepatic encephalopathy, most likely secondary to a congenital portosystemic vascular anomaly (PSVA).

Further diagnostic tests included determination of clotting times (normal), BSP retention (5% at 30 min), 2 h postprandial serum bile acid measurements (greatly elevated; 109.5 μmol/L), and serum uric acid measurements (normal). A cranial mesenteric venogram was performed and a large extrahepatic portosystemic shunt was identified. As contrast material was excreted by the kidneys, it was noted that the left kidney, left renal pelvis, and proximal one-half of the left ureter were greatly dilated (Figure 1). The renal diverticula were within normal limits, which was consistent with acute onset of hydronephrosis. A radiolucent filling defect suggestive of a urolith was seen in the midsection of the left ureter.

Surgical correction of the portosystemic shunt, a liver biopsy, and removal of the urate urolith were performed. The shunting vessel was identified as the gastrosplenic vein and was completely ligated. Postligation portal pressures were within normal limits. Urine was aspirated from the left renal pelvis and ureter. A calculus was palpated in the left ureter and an incision was made proximal to the calculus. A single, pyramidal-shaped, yellow-green, 5 × 7 mm calculus was removed from the ureter. The ureter was flushed proximally and distally with saline to remove accumulated sediment and crystals, and the ureter was closed with interrupted sutures of 5–0 polyglactin 910 (Vicryl, Ethicon Inc., Sommerville, New Jersey). There were no palpable calculi in the urinary bladder.

On histopathological examination of a liver biopsy, changes compatible with a portosystemic vascular anomaly were identified (portal fibrosis, vacuolation of centrilobular hepatocytes, and paucity of portal veins). There were numerous ammonium urate and tyrosine crystals, and occasional rod-shaped bacteria in the urine aspirated from the left renal pelvis and ureter. Quantitative analysis of the urolith revealed 88% ammonium acid urate composition.

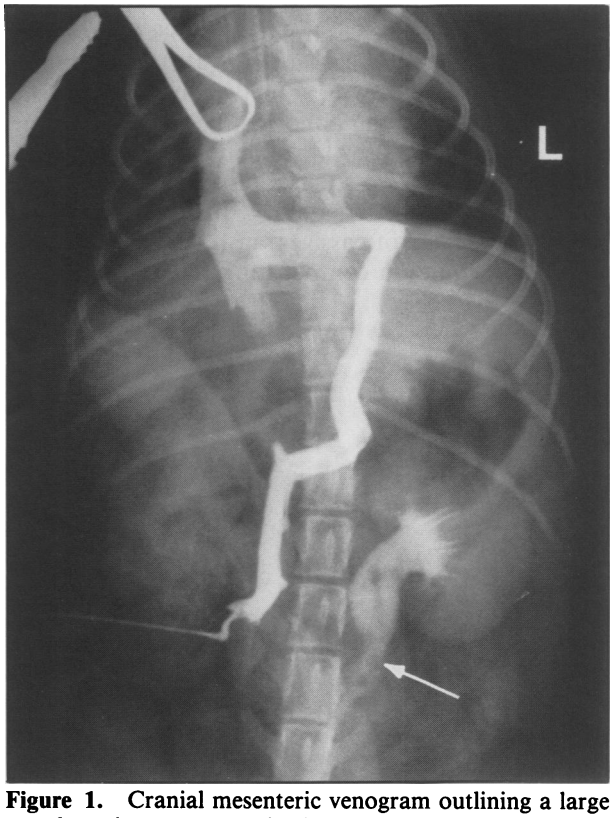

Figure 1. Cranial mesenteric venogram outlining a large extrahepatic portosystemic shunt (the gastrosplenic vein). Radiopaque contrast material injected for the venogram has been excreted by the urinary system, outlining the distended left kidney, renal diverticula and pelvis, and ureter. A urolith is present in the ureter (arrow).

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Trimethoprim-sulfadiazine (Tribrissen, Cooper-Wellcome, Kansas City, Missouri) (15 mg/kg PO q12h) was administered to the dog for 2 weeks.

One month after surgery, a CBC, serum biochemical profile, urinalysis, serum ammonia and uric acid measurements, and a 2 h postprandial serum bile acid measurement were within normal limits. An excretory urogram revealed normal size and function of both kidneys, with a marked reduction in diameter of the left hydroureter. The dog was not returned for a two-month postsurgical reexamination and was lost to follow-up.

A high prevalence of ammonium urate uroliths has been reported in dogs that have a portosystemic vascular anomaly (1,2). Any pathological or physiological process that results in enhanced excretion of ammonium ion and uric acid results in a predisposition to the formation and precipitation of ammonium urate (3). Several disease processes and physiological disorders which result in urate lithogenesis have been documented in man and domestic animals. These include primary and idiopathic gout, inborn errors of purine metabolism, excess dietary purines, hyperuricosuria, renal insufficiency, and hepatic insufficiency (3).

The predisposition of dogs with PSVA to develop ammonium urate uroliths presumably reflects the metabolic derangements associated with hepatic insufficiency. Dogs with PSVA may have increased serum levels of ammonium ion (4,5) and uric acid (6), which results in increased urinary excretion of these substances. This may result in the formation of ammonium urate uroliths. Among dogs that have a portosystemic vascular anomaly, Yorkshire terriers are predisposed to the formation of ammonium urate uroliths (2,7).

While exact modes of urate lithogenesis remain unidentified, enhanced urine uric acid concentrations appear to be a major predisposing factor in human patients (3). The role of uric acid in urate lithogenesis in dogs is less certain. Uric acid levels in dogs with PSVA have been reported to be either normal (8) or elevated (6). While pre- and postsurgical serum uric acid measurements were within normal limits in our dog, postsurgical levels were lower than the presurgical levels. However, normal values for canine serum uric acid levels have not been established, and human serum uric acid assays may not be accurate for dogs.

Other factors that may contribute to the formation of ammonium urate uroliths are the poor solubility of uric acid and the presence of urease-producing microorganisms in the urinary tract. While uric acid is itself poorly soluble in aqueous solutions, ammonium urate is the least soluble of the uric acid salts (9). Urease-producing microorganisms may increase the formation and precipitation of ammonium urate by 1) increasing the local production of ammonium ion, 2) alkalinizing the urine, thereby further reducing the solubility of ammonium urate, and 3) increasing the coprecipitation of other mineral species such as struvite (10). Since rod-shaped bacteria were found in urine aspirated from the affected kidney and ureter, it is possible that urease-producing microorganisms were present and could have contributed to the formation of the urolith. Identification of the organisms by bacterial culture in our dog was not attempted.

The radiographic appearance of urate uroliths depends upon size and mineral composition. Pure uric acid uroliths (which occur primarily in humans) are typically radiolucent (11). Most ammonium urate uroliths in dogs are moderately radiodense (2), but they are less radiodense than struvite uroliths (6). Urate uroliths that are small, radiolucent, or located in the kidney may not be visualized by survey radiography. Positive contrast radiography may be necessary to identify the presence of these uroliths, as in this dog.

This case is of interest because of the unusual finding of hydronephrosis and hydroureter caused by an ammonium urate urolith in a dog with a PSVA. Although renal, vesicular, and urethral ammonium urate uroliths have been described (6), we could not find a report of ureteral obstruction by a urate urolith in a dog with a PSVA. The pyramidal shape of the urolith suggests that the urolith formed in the left renal pelvis and was dislodged into the ureter, with resultant hydronephrosis and hydroureter. Prompt surgical removal of the urolith resulted in restoration of normal renal function. Until the enlarged left kidney and ureter, and the filling defect in the ureter, were outlined by the radiopaque contrast material injected for the cranial mesenteric venogram, there was no reason to suspect abnormalities in the urinary tract. Radiopaque contrast material injected during a cranial mesenteric venogram may be used to screen the urinary tract for uroliths and associated complications in dogs with suspected portosystemic vascular anomalies. CVJ

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