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Feline Urologic Syndrome in the Male Cat

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Incidence

Feline urologic syndrome (FUS) can be defined as dysuria and hematuria in cats of both sexes.¹ The disease can be manifested by the following syndromes: 1) cystitis, 2) urethritis, 3) urethral plugs composed of mucoprotein, 4) mineral calculi, 5) urethral obstruction.² FUS is estimated to affect approximately 1% of the cat population of the United States, accounting for up to 10% of the total number of male cats seen in veterinary practice. Only cases of upper respiratory infection and cat bite abscesses have higher incidence.^{2,3} There is both an age and seasonal predisposition. Cats between the ages of one and three years make up 79% of the FUS cases. The months December through March have the highest number of cases, speculated to be due to decreased exercise of cats confined during colder weather.³ There is a higher incidence of FUS in female cats than male cats.² Females more commonly show the cystitis or urethritis components of the syndrome; males are more susceptible to urethral plugs, calculi, and urethral obstruction.⁴ In the following paper, only that syndrome as seen in the male cat will be discussed. Etiology (both primary and secondary), clinical and laboratory findings, medicinal therapy, and preventive measures for FUS will be discussed.

Etiology

The cause of feline urologic syndrome in the male cat has been a subject of considerable research. The formation of mineral and matrix urethral plugs and calculi involves at least four factors.^{2,4} First, the presence of a nucleation center for deposition of minerals may be necessary. A variety of substances including viruses,

bacteria, sloughed epithelial cells, casts, etc., may serve as such a center. In experimentally produced disease, three viruses have been implicated: a) Picornavirus—designated Manx calicivirus—thought to be the triggering agent for b) Syncytium-forming Myxovirus—once activated, this virus is speculated to produce cellular damage within the urinary tract leading to calculi formation; c) Herpesvirus—it is hypothesized this virus causes the production of a unique, unidentified protein found in the urine and urethral plugs of affected cats, and this protein may serve as a matrix to aid in the conglomeration of struvite crystals.⁵ However, in naturally occurring cases of FUS, viruses have rarely been found either by electron microscopy or viral culture, and it appears that if they are a predisposing cause for FUS, they do so primarily by providing a nucleation center on which calculi can form.⁴

In addition to the matrix nucleation concept, two other theories have also been suggested for stone formation. The precipitation-crystallization theory incriminates supersaturation of urine with crystalloids as the primary factor in precipitation and subsequent growth of calculi. The inhibitor absence theory suggests the absence in urine of an inhibitor of calculi formation as the primary factor in stone formation.⁸

Bacteria also may increase material available for a nucleation center by themselves or by causing urinary tract damage or inflammation with resulting debris. In one study, only one of 38 cats had a positive bacterial culture of urine (greater than 10^5 organisms per ml of urine).¹ In a similar study, low numbers (less than 10^5 organisms per ml) of *Staphylococcus*, *Pasteurella*, *Corynebacterium*, *Clostridium*, *E. coli*, and *Bacillus* were isolated from ten of 48 FUS-affected cats.⁶ Another study tested the hypothesis that FUS may be caused by a horizontally transmitted infective agent, but found no difference in

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prevalence of the disease among cats living in the same household with an affected cat as a control group.⁷

The second factor contributing to the development of calculi is urine pH. The solubility of the calculogenic substances calcium, magnesium and phosphate is greatly increased in a urine pH less than 6.6. Urine pH can be raised, and subsequent formation of calculi can be increased in urinary tract infections from urease-producing bacteria such as *Staphylococcus*, *Pseudomonas*, and *Proteus* (these organisms split urea into carbon dioxide and ammonia, a weak base).⁴

The third factor important in calculi formation is the formation of a concentrated urine. This can be due to a decreased water intake or infrequent urination. The more concentrated the urine, the more concentrated the crystal-forming substances, and the greater the chances for calculi formation. Infrequent voiding also allows more time for calculi formation to occur, and promotes an increased urine pH.⁴

The fourth and final factor necessary for calculi formation is a high concentration of magnesium or phosphorus in urine ultimately derived from high dietary levels.² More than 97% of the calculi occurring in cats are struvite crystals — $\text{NH}_4\text{PO}_4\text{Mg}\cdot 6\text{H}_2\text{O}$. Struvite crystals are a normal finding in the urine of cats. However, studies by at least three groups of investigators indicate that a diet high in magnesium, high in phosphorus, low in chloride, or with a calcium:phosphorus ratio less than one, predisposes cats to urolithiasis.⁸ One study induced urethral obstruction in male cats by feeding a diet containing .75% magnesium and 1.6% phosphorus on a dry matter basis.^{8,10,11} Studies also show that dietary calcium helps prevent calculi formation when either dietary magnesium or phosphorus are low, but not when both are high. In fact, when both are high, increasing dietary calcium enhances calculi formation.⁴ These factors become important later in formulating a preventative diet for the FUS-stricken cat.

There are several secondary predisposing factors that have been described. In general, cats as a species are more susceptible to urologic problems due to their nature of having prolonged periods between urinations. Some cats urinate less than once a day. This concentrates urine and minerals and promotes calculi formation. The male cat especially is susceptible to urethral obstruction due to his anatomy. His

longer, narrower urethra is more conducive to blockage than that of the female.³ Although any portion of the urethra may obstruct, the penile urethra is the most common area due to its smaller diameter.⁵

In addition, researchers are now speculating that the disseminated prostate glands found in the post-prostatic urethra of male cats may produce at least some of the matrix contributing to the formation of urethral plugs. These glands produce mucus, and may possibly be a component of local host defenses of the lower urinary tract. It is hypothesized that the secretory activity of these glands may be enhanced by viral infections or other pathogenic stimuli, creating a sequence of events analogous to obstruction of nasal passages with excessive mucus production caused by viral rhinitis. Crystals, cells, and other substances may then be trapped in an organic matrix. At the present time, the role, if any, of disseminated prostate glands in FUS is unknown.¹²

Studies comparing castrated versus intact cats show no difference between the two groups in incidence of FUS. There is also no correlation between age at castration and onset of FUS, as cats castrated before puberty have the same urethral diameter as cats castrated post-pubertally.¹¹ However, researchers did note the tip of the penis in castrated cats contained more fibrous tissue than in noncastrated cats, possibly decreasing the potential of the castrated cat's urethra to dilate.⁵ Castrated cats have a longer average survival time and lower mortality rate after the onset of urolithiasis than intact cats (3.65 years versus 2.6 years).¹³

There also appears to be no correlation between the incidence of FUS and body weight, weight gain, or body condition, although obesity may indirectly predispose to the syndrome by decreasing physical activity.^{4,13}

As previously mentioned, diet fed to the cat may be an important predisposing factor. Diets with high levels of magnesium and phosphorus may be conducive to calculi formation. The form of the diet is also important. Dry diets appear to predispose to the syndrome more than canned diets, because on a dry matter basis most dry diets contain less available energy than canned diets per gram. Therefore, the cat must ingest a greater amount of the dry diet and associated minerals to meet energy needs.¹³ In a study of 101 male cats and 101 control cats it was found cats eating greater than one half of their diet in dry form had a

significantly higher relative risk for urethral obstruction than those on a canned diet, and the risk continued to rise with cats eating greater than 75% dry food and those eating dry food exclusively.⁷

In summary, the cause of the feline urologic syndrome in the male cat appears to be multifactorial. A specific organism directly responsible for the calculi or urethral plugs in naturally occurring FUS has yet to be identified. However, it has been proven that presence of a nucleation center, urine pH, urine concentration, and dietary levels of magnesium, phosphorus and calcium are of prime importance in causing the syndrome. Other contributing factors include the anatomy and urination behavior of the male cat, his level of physical activity, and form of diet fed to him.

Clinical Findings

The clinical signs of the feline urologic syndrome can be classified according to the duration and urgency of the disease. In the early stages, dysuria and/or hematuria are the predominant symptoms. Owners often complain of a housebroken cat suddenly urinating in unusual locations. The cat will squat and strain—passing no urine at all or perhaps only small amounts of blood-tinged urine. The urine will characteristically have a strong ammonia-like odor. Owners will also frequently complain of anorexia in the cat and note him excessively licking at his penis.⁴

If the disease progresses to become severe or the urethra becomes blocked, clinical signs can progress to include vomiting, depression, dehydration and azotemia. Abdominal palpation will distinguish a cat with urethral obstruction from a nonobstructed one; a nonobstructed cat will likely have a small, easily expressible bladder while an obstructed urethra will result in a firm, distended, painful bladder.³

If the urethra remains blocked, coma, hypothermia, and ultimately death follow within 48 hours.⁴ Death will be due to dehydration, hyperkalemia, metabolic acidosis, and an accumulation of metabolic waste products.⁵ The bladder may also rupture—usually a fatal consequence due to the poor surgical risk of such a cat. By the time a cat reaches the comatose stage, irreversible damage may have already been done to the kidneys from azotemic changes and abnormal back pressure.

Typical laboratory findings would include the following: a urinalysis commonly shows

proteinuria and hematuria; there may be a mild to moderate glucosuria due to a stress hyperglycemia; there is usually a concentrated urine specific gravity, and struvite crystals are a common finding; a wide range of urinary pH's are found with FUS cats, and most urine cultures are negative for bacteria.^{3,5}

A typical hemogram would be that of a stress-induced neutrophilia, lymphopenia, and eosinopenia. Blood urea nitrogen concentration will be variably elevated depending on the duration and severity of the obstruction, and creatinine levels are usually elevated. Blood pH often indicates a metabolic acidosis due to a decrease in renal clearance of hydrogen ions, and a blood pH less than seven is usually lethal. In 7 to 10% of cats showing clinical signs, there is a hyperkalemia. At potassium levels greater than 7 mEq/liter, cardiac conduction disturbances and generalized muscle weakness begin to be observed. Death due to cardiac arrest may occur when potassium levels exceed 10 mEq/liter. Also, metabolic acidosis aggravates hyperkalemia as the kidneys retain potassium while attempting to excrete excess hydrogen ions.⁵

Therapy

Treatment of the FUS-stricken cat can be classified by the stage of the disease the cat is in. According to the majority of authors, therapy for the nonobstructed cat should include the following: antibiotics need not be routine, but if used, broad-spectrum drugs which are excreted in the urine should be administered for five to ten days; urinary acidifying agents may be given; the cat should also be fed a low ash, no milk diet; extra water may be mixed with each meal to increase water intake; a recheck examination should be scheduled after the initial episode, and if the cat is progressing well the antibiotics can then be discontinued. Ideally, rechecks should also be scheduled for several times during the year so the cat's condition can be monitored. Appropriate preventative measures (to be discussed later) can be started then if necessary.¹⁴ Some authors do not suggest the use of antibiotics on nonobstructed cats unless they have a positive urine culture. In one study using chloramphenicol or a placebo for the treatment of 20 cats, it was concluded that no benefit was derived from antibiotic treatment. Clinical signs resolved in 70% of cats within five days regardless of treatment, and a 39% rate of recurrence of clinical

signs within 18 months was also noted regardless of treatment used.¹

Treatment for the obstructed cat is considered an emergency procedure, and the re-establishment of urine flow should receive top priority.¹⁵ Anesthesia is usually necessary to relieve the urethral obstruction. It is suggested to avoid drugs that are excreted in active form by the kidneys and therefore require functional kidneys for inactivation—such as ketamine HCl.³ The first step in attempting to dislodge the plug in the penile urethra should be to gently massage the penis between the thumb and fingers. Plugs in the abdominal or pelvic urethra may be dislodged by massaging the urethra per rectum. If this technique is not successful in dislodging the obstruction, urinary catheterization and reverse flushing will be necessary. The correct technique is the following: The penis and prepuce should be cleansed with warm water. A sterile polyethylene tom cat catheter (number 3½ French by Sherwood, Brunswick or Portex) should be lubricated with a sterile aqueous coating.¹⁴ “Meticulous aseptic and feather touch technique” should be utilized to prevent damage to the delicate tissues of the urethra and bladder as the catheter is inserted in the urethra until the point of blockage.¹⁵ At this point a large amount (several hundred mls) of lactated Ringers solution (preferred by some authors because of its low cost) or sterile saline should be flushed into the urethral lumen and allowed to reflux out of the external urethral orifice. As the catheter is advanced to the bladder, the obstruction material should be gradually dislodged and flushed around the catheter and out of the urethra.¹⁵ Excessive pressure should not be used as it may cause bladder trauma and reflux of potentially infected urine into the ureters and the renal pelvis.¹³ If necessary to maintain urine flow after relief of the obstruction, a catheter can be sutured to the prepuce and the bladder completely emptied with a syringe. Smooth muscle relaxants are not consistently effective in aiding catheterization, as the preprostatic urethra is surrounded by smooth muscle; but distal to the prostate there is an inner layer of smooth muscle and an outer layer of skeletal muscle.¹⁵

If the obstruction has yet to be relieved, cystocentesis may be necessary. The skin area should be surgically prepped. A 22-gauge one and one-half inch needle with either a large syringe or a two or three-way stopcock should

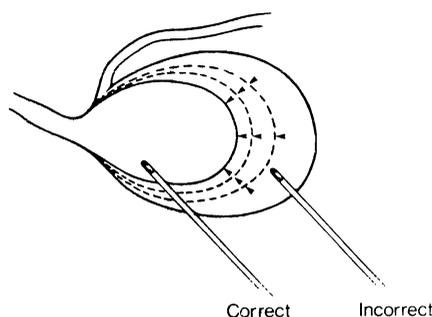


Figure 1. Illustration of correct and incorrect sites for insertion of a needle into the bladder.¹⁵

be inserted in the ventrolateral bladder at a 45° angle cranial to the junction of the bladder with the urethra. This location should minimize the chance of trauma to the ureters and major vessels and allows the needle to stay in the lumen as the bladder decreases in size (Figure 1).¹⁵

Typical post-obstruction care involves leaving the catheter in place 12 to 72 hours. Some researchers feel it is unnecessary to leave an indwelling catheter if the cat is not depressed and a full stream of urine is elicited upon catheterization.³ However, a urinary bladder may become atonic if obstruction has been present an extended length of time, and an indwelling urinary catheter maintains an empty bladder lumen allowing the detrusor muscle to regain tone.¹⁵ The cat should be hospitalized while the catheter is indwelling, and observed for reobstruction for 24 hours following catheter removal. During this time, diuresis should be promoted using lactated Ringers solution intravenously or subcutaneously. To decrease the frequency of reobstruction, a goal would be to produce a urine with a specific gravity less than or equal to 1.030.¹⁵ A moderate to massive diuresis can normally be expected following relief of obstruction due to a proximal tubular defect in resorption of sodium and solute diuresis resulting from retained metabolites during obstruction.³ Urecholine^a (Bethanechol Cl) is a parasympathomimetic drug which stimulates the bladder to contract and promotes voiding of residual urine, and can be given to patients

^aUrechole, Merck, Sharp & Dohme division of Merck & Co., West Point, PA.

with a patent urethra and decreased bladder tone. Systemic antimicrobials should also be started to decrease the possibility of a bacterial urinary tract infection, and continued 14 to 21 days.¹⁶ When the cat is sent home, further preventive measures should be vigorously instituted.

The aftercare of uremic cats following relief of urethral obstruction has a few more points to be taken into consideration than with the cat obstructed of short duration. Monitoring of body temperature is important, and a heating pad may be necessary for cats with subnormal temperatures.¹⁴ Dehydration, metabolic acidosis and hyperkalemia will be the most life-threatening problems. Fluid deficits, dehydration, and maintenance requirements should be corrected for using intravenous D5W (preferable) or lactated Ringers solution.¹⁵ Metabolic acidosis should also be assessed using blood pH, or such symptoms as urine pH less than 5.5; increased blood urea nitrogen and creatinine, and severe depression are suggestive of acidosis. The appropriate amount of sodium bicarbonate should be administered slowly over a 48-hour period.¹⁵ Most hyperkalemia patients are successfully managed by removing the obstruction and rehydrating, but severe hyperkalemia can be treated with regular insulin given only once, followed by dextrose intravenously to cause potassium to passively follow glucose intracellularly.¹⁵

Two therapeutic contraindications should also be kept in mind in the aftercare of the uremic cat. Glucocorticoids should not be administered, as they induce gluconeogenesis and cause more protein by-products that the kidney must eliminate. Also, urinary acidifiers should not be used at this time as they will aggravate metabolic acidosis.¹⁵

Prevention and Management

The prevention and management of FUS is a very important part of any discussion of the disease. Clients should be made to understand that FUS can be a recurrent disease. With certain dietary changes and medication, recurrence is uncommon; without them, the recurrence rate can be as high as 50 to 75%.¹⁷

The prevention of the syndrome is directed towards lessening the contribution of each of the factors necessary for calculi formation. To decrease the material available for a nucleation center, urinary tract infections should be treated as determined by culture and sensitiv-

ity for two to three weeks. A urine culture should be run one week after the termination of the antibiotics, and if it is positive, antibiotic therapy re-instituted.⁴ One researcher advises no systemic antibiotics should be necessary unless a positive urine culture is obtained or a urinary catheter has been placed.¹⁷

The urine concentration of calculi-forming minerals can be decreased by 1) decreasing dietary intake by feeding rations low in magnesium and phosphorus that still meet the cat's requirement and 2) increasing urine volume, therefore, also increasing the frequency of urination and decreasing the amount of time available for calculi to form. The optimum dietary mineral concentrations for prevention of FUS are less than 0.1% magnesium and less than 1.0% phosphorus in the dry matter. Based on currently available research data, the following diet recommendations can be made. Dry rations, as discussed earlier, appear to predispose to FUS, and all commercial dry and soft moist rations, except Prescription Diet C/D and Iams, contain magnesium and phosphorus levels higher than considered optimum for the prevention of FUS. If a dry ration is necessary to use, these two should be recommended. Of 48 commercial canned cat foods analyzed, four consistently met requirements of an optimum diet for FUS prevention and should be recommended. These include: 1) Prescription Diet C/D, 2) Heinz Nine Lives Liver, 3) Friskies Buffet Liver and Beef, 4) Friskies Buffet Turkey and Giblets. However, Heinz Nine Lives Liver is deficient in calcium and may cause bone demineralization due to nutritional secondary hyperparathyroidism; therefore, it should not be fed for an extended period of time.⁴ Also, a homemade diet has been formulated that meets the requirement for FUS prevention (Figure 2).²

Figure 2. Homemade Low Magnesium/Phosphorus Ration for Cats²

1 lb regular ground beef, braised lightly and retain the fat

¼ lb liver, uncooked or braised lightly

1 cup cooked rice

1 teaspoon cooking oil

1 teaspoon limestone

2-3 oz of water can be added during cooking. Combine all the ingredients.

Yield: 1¾ lb.

Feed: ¼ to ½ lb/cat/day

Contains 0.5% phosphorus and 0.05% magnesium.

Urine volume and frequency of urination may be increased by adding table salt (sodium chloride) at one-half teaspoon per day to the ration. This not only increased the water intake, but the chloride in the urine provides an anion to displace phosphates, carbonates, and sulfates from calcium and magnesium, causing the formation of soluble rather than insoluble salts.⁴

Another step in FUS prevention is altering urinary pH to make it unfavorable for mineral crystallization—i.e., acidifying the urine. Phosphate calculi form best at a pH of seven or greater, and their solubility increased greatly at a pH less than 6.6. An acid urine is more bacteriostatic than an alkaline urine, and enhances the effect of urinary antibiotics. Studies show that of the commonly used urinary acidifiers, methionine, ascorbic acid, and sodium acid phosphate have no effect on urine pH in the cat. In contrast, ammonium chloride fed once per day maintained urine pH at 5.8 to 6.1 throughout the entire day. This low pH was maintained even in the face of the normal “postprandial alkaline tide,” a rise in urine pH from approximately 6.1 prior to eating to 7.2 in the first four hours after eating due to meal-induced gastric acid secretion. This temporary postprandial increase in urine pH may allow calculi to form even though urine is acidic the rest of the day.⁴

Although methionine is not regarded by some to be an effective urinary acidifier, it does increase urinary excretion of sulfate which acts to displace phosphate in struvite calculi. Therefore, although the addition of 1% methionine to the diet will not prevent obstruction in cats fed calculi-producing diets, it will significantly prolong the time before obstruction occurs.¹⁷ A product frequently used is Urozeze FUS,^b and it contains 1.25 grams ammonium chloride and 1.25 grams methionine per teaspoon. Recommended dosage is $\frac{3}{4}$ to one teaspoon mixed well in food, given once per day.² If mixed well in the food, palatability won't be affected; if not well mixed, food intake may be decreased.¹⁷

For the cat never affected with FUS (this includes 99% of all cats), there is no need to either restrict them from dry rations, or feed them rations low in magnesium or with added ammonium chloride. A good national brand commercial cat food in dry, soft moist, or canned form will be sufficient for such cats.¹⁷

^bManufactured by Daniels Pharmaceuticals, Inc.

In addition to these changes, a few other recommendations can be made to clients to help their cats. As FUS can be a recurring disease, owners should learn to observe their cat's micturition habits, urine color, and possibly should be taught to palpate the bladder so they will be able to detect abnormalities if they should occur.¹⁸ Clients should always supply their cat with a clean litterbox to encourage him to urinate frequently, and reduce the amount of litter used in the box to prevent urethral trauma from excess litter particles.³ The cat should be provided with free-choice, clean, fresh water that is changed daily, and he should be encouraged to exercise.⁴ In addition, Washington State University research has indicated that vaccination with Norden's Felimmune CVR product seems to decrease the incidence of FUS.³ This may be another reason to encourage routine immunization of cats.

Conclusion

Feline urologic syndrome is one of the major diseases affecting the cat and is of prime importance in small animal veterinary practice. One study has cited a 1.2% mortality rate for FUS, and noted that with proper diet and medication, this rate could easily be reduced to zero.¹⁴ In another study, in a six-month period, 29 of 101 cats receiving no management reobstructed. With management, only two of 93 other cats reobstructed during a three-year period.⁴ Such figures indicate that FUS is a disease that can be controlled with a combination of good veterinary and cooperative client care.

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BOOK REVIEW

Animal Pain Perception and Alleviation, Edited by R. L. Kitchell and H. H. Erickson, American Physiological Society, Bethesda, Maryland, 1983.

This book is a collection of papers from a symposium on pain in animals, which was held in conjunction with the 66th Annual Meeting of the Federation of the American Societies for Experimental Biology (FASEB) in New Orleans, Louisiana, April, 1982. The book is divided into two parts: the perception of pain and the alleviation of pain. The pain perception section contains current information on peripheral, spinal, and supraspinal pain mechanisms in animals, anatomical pathways and pain control systems, segmental neurophysiological mechanisms, stimulation-produced analgesia, behavioral procedures for the assessment of pain, pain assessment during surgical manipulations and the phylogenesis of pain expression in animals. The section on the alleviation of pain is brief. It contains a discussion of important species differences in the absorption and biotransformation of drugs used to alleviate pain. Also included are papers on analgesic drug evaluation procedures in horses and a summary of common agents used to control pain in dogs and cats.

Most of the papers were written by authorities in their respective fields. Interestingly, even

though the book is devoted to a discussion of animal pain, only a few of the authors are veterinarians. The book is well organized, easy to follow, and generally easy to read. Most of the authors have presented adequate research data that reflected the extent of our knowledge of pain mechanisms at the time of the symposium. This was particularly true for the chapters on descending control systems and stimulation-produced analgesia. The chapters on general anesthesia and control of pain in cats and dogs contain useful summaries of information that can be found in most modern veterinary pharmacology textbooks but add little information on new methods or drugs to alleviate pain.

This book should be read by every scientist doing animal research in which procedures are employed that are painful or even potentially painful. In addition, this collection of papers should be of interest to various regulatory officials who have to be concerned about the health and welfare of animals used in numerous research and educational facilities. The topic of animal pain should be of interest to all practicing veterinarians; therefore, this book may be of value to those practitioners who are well-versed in neuroanatomy and neurophysiology.

—DR. D. D. DRAPER