Clinical Survey and Selection of Therapeutic Approach for Emergent Feline Urological Syndrome

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Abstract: The management of cases of the emergent feline urological syndrome (FUS) is described with particular reference to urethral obstruction in the tomcat. Treatment of the obstructive episode, medical therapy in the post–obstruction period and the prevention of recurrence of blockage in the longer term are discussed. The target of the present investigation was rapid confirmation of diagnosis of feline urological syndrome with epidemiological studies and to choose the suitable life saving therapeutic regimen. Also, the important aim was prevention of recurrence of such cases. The present study was carried out on forty seven tomcats (42 clinically diseased tomcats and 5 apparently healthy tomcats). Clinical manifestations were stranguria (n= 23), bloody urine (n= 23) and frequent licking of the urinary opening (n= 17), frequent attempts to urinate end with failure (n= 17), ishuria (n= 19), excessive salivation (n= 15), increased respiratory and pulse rates and vomiting (n= 13). Clinical examination was revealed severely distended urinary bladder and abdominal tenderness. Ultrasonographic examination revealed severe distension of urinary bladder, turbidity inside urinary bladder (floating debris) and dilation of renal pelvis (Hypoechogenicity). Therapeutic approaches in order were either gentle manual compression of the bladder with milking of penile urethra or catheterization or cytocentesis followed by Perineal urethrostomy. It was concluded that confirmation of emergent feline urological syndrome by rigid urinary bladder during palpation. FUS was aggravated when vomiting was presented and accompanied by dilation of renal pelvis detected by ultrasonography. The most selected therapeutic regimen by the present investigation was catheterization (64.3%).
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1. Introduction

Problems that affect a cat's lower urinary system often prevent the bladder from emptying correctly or may even cause fatal blockage of the urethra, the tube connecting the bladder to the outside of the body. Very often the culprit is Feline Lower Urinary Tract Disease (FLUTD). Once called Feline Urologic Syndrome (FUS), FLUTD is not merely one problem, but a collection of clinical symptoms that may have more than one possible cause. Symptoms of FLUTD include frequent or painful urination, bloody urine and frequent licking of the urinary opening. One key to treating FLUTD is to determine the root cause, which may include bladder stones, urinary tract blockage, infection or cancer. If the cause of these symptoms cannot be determined, the cat is considered to have bladder inflammation (cystitis) as mentioned by **Osborne et al. (1992**).

FLUTD is rarely diagnosed in animals younger than one year; the average age is typically four years. Male cats are generally more prone to urethral blockages because of their narrower urethras **(Leib and Monroe, 1997)**.

Untreated FUS can cause partial or complete obstruction of the urethra, preventing a cat from urinating. This is a medical emergency that can very quickly lead to kidney failure and/or rupture of the bladder, and can prove fatal if the obstruction is not relieved right away **(Gaskell et al., 1978)**.

There are two major types of crystals. The first was struvite crystals which form in urine with elevated pH (>6.8, even more likely with continued levels above 7.0) while the second was calcium oxalate crystals (which form in too acidic urine <6.0). Struvite crystals can be addressed with diet, however if your cat has oxalate crystals, that's a lot trickier to deal with. There are no cat-safe herbs for oxalate crystals so please beware of herbal formulas *e.g.* those labeled "stone-breakers". Homeopathy (meaning a properly chosen single remedy, not a combo product) is the only safe non-surgical option for oxalate stones. Before acidifying diets started getting "prescribed", oxalate crystals were less commonly seen. Struvite crystals are still far more common **(Leib and Monroe, 1997).**

The target of our investigation was rapid confirmation of diagnosis of feline urological syndrome with epidemiological investigations and to select the suitable life saving therapeutic regimen. Also, the important aim was prevention of recurrence of such cases.

2. Materials and methods

Animals:

A total number of 47 cats (2.7-10.0 years-old, Persian tom cats) admitted to the clinic of faculty of veterinary medicine, Cairo University and to a private veterinary clinic in Giza governorate. On the basis of history and clinical presentation, the cats were divided into 2 groups; group (1); a group of 5 apparently healthy cats (2.9- 7.3 Ys old) and group (2); a group of 42 clinically diseased cats (2.7- 10 Ys old).

Clinical evaluation:

Age, gender, breed, feeding, history of catheterization, respiratory rate, pulse rate, rectal temperature of the cats of the study was recorded before treatment. All cats were thoroughly investigated and clinically examined by abdominal palpation and tactile percussion before treatment according to the method described by **Kelly (1984)**.

*Ultrasonographic examination:

Ultrasonography was performed after 24 hrs fasting before therapeutic approach. The examined cats were positioned in dorsal recumbency. Cranial ventral abdomen were clipped and sheaved then covered with coupling gel. Transverse and longitudinal scans were taken using Pie-Medical Scanner (Maastricht, Netherlands) and sector transducer with alternating frequency of 5.0-7.5 MHz according to the method described by **Nyland et al., (1989)**.

Therapeutic regimen:

Therapeutic approaches in order were either gentle manual compression of the bladder with milking of penile urethra or catheterization or cytocentesis followed by Perineal urethrostomy. Recording of the period of post-obstructive medical treatment were done and diet change by prevention of the offending dry food (according to **Leib and Monroe, 1997**).

Statistical analysis was performed by statistical Package for Social Sciences (SPSS). Mean and standard deviation are descriptive values for quantitative data. ANOVA (Analysis Of Variance) was used for testing means of more than two groups by computer program according to the method described by **Irwan (1996).**

3. Results

All recorded cases were Persian tomcats of age between 2.7- 10 years old. Feeding of 37 out of 42 tomcats (88.1%) were commercial dry food. Seventeen tomcats were castrated (17 out of 42, 40.5%). History of clinical signs said by owners were frequent trips to the litter box, frequent urination, urination in very small amounts, blood in the urine, urination outside the litter box, and straining or crying (moaning) during urination. Clinical manifestations in advanced cases were stranguria (n= 23), bloody urine (n= 23, Fig. I, 1 1and 2) and frequent licking of the urinary opening (n= 17), frequent attempts to urinate end with failure (n= 17), ishuria (n= 19), excessive salivation (n= 15), lethargy, vomiting (n= 13) and presence of toothpaste like struvite after squeezing of urinary bladder or catheterization (Fig. I, 3). There were significant increases in respiratory and pulse rates (table 1).

Table (1): General clinical examination of Group (1) and Group (2), Mean values ± standard deviation

Parameters	Group (1)	Group (2)
Respiratory rate	29.6 ± 1.8	49.11 ± 7.94
Pulse rate	89.8 ± 5.36	152.45 ± 10.97
Rectal temperature (°C)	38.66 ± 0.27	38.3 ± 0.24
Mucous membranes	Very faint rosy red	Very faint rosy red
Superficial lymph nodes	Free	Free

Results of independent samples t-test showed that there was a significant increases of respiratory and pulse rates at P value <0.05

Results of Clinical examination:

Clinical examination was revealed severely distended urinary bladder and abdominal tenderness.

Results of Ultrasonography:

2- Group (1): Apparently Healthy cats:

Sonogram of the kidney and urinary bladder revealed hypoechoic renal cortex relative to the adjacent spleen, which provided an acoustic window for examination of the kidney. The renal pelvic recesses contained fat and appeared as hyperechoic regions in the center of the kidney. The renal medulla was hypoechoic relative to the renal cortex. In the transverse sonogram the renal hilus was hyperechoic because of the fat that is present. The renal pyramid was seen as a V-shaped hypoechoic structure extending into the hyperechoic hilar fat. Retroperitoneal fat provided a bright line that outlined the kidney (Normal kidney). Urinary bladder scan revealed anechoic urine lined by double hyperechoic lines (double lamellate).

2- Group (2): Clinically diseased cats before treatment

Ultrasonographic examination revealed severe distension of urinary bladder, turbidity inside urinary bladder (floating debris) by agitation and

dilation of renal pelvis (Hypoechogenicity, 13 cases of vomiting) and hypoechoic renal cortex (Fig. I, 4).

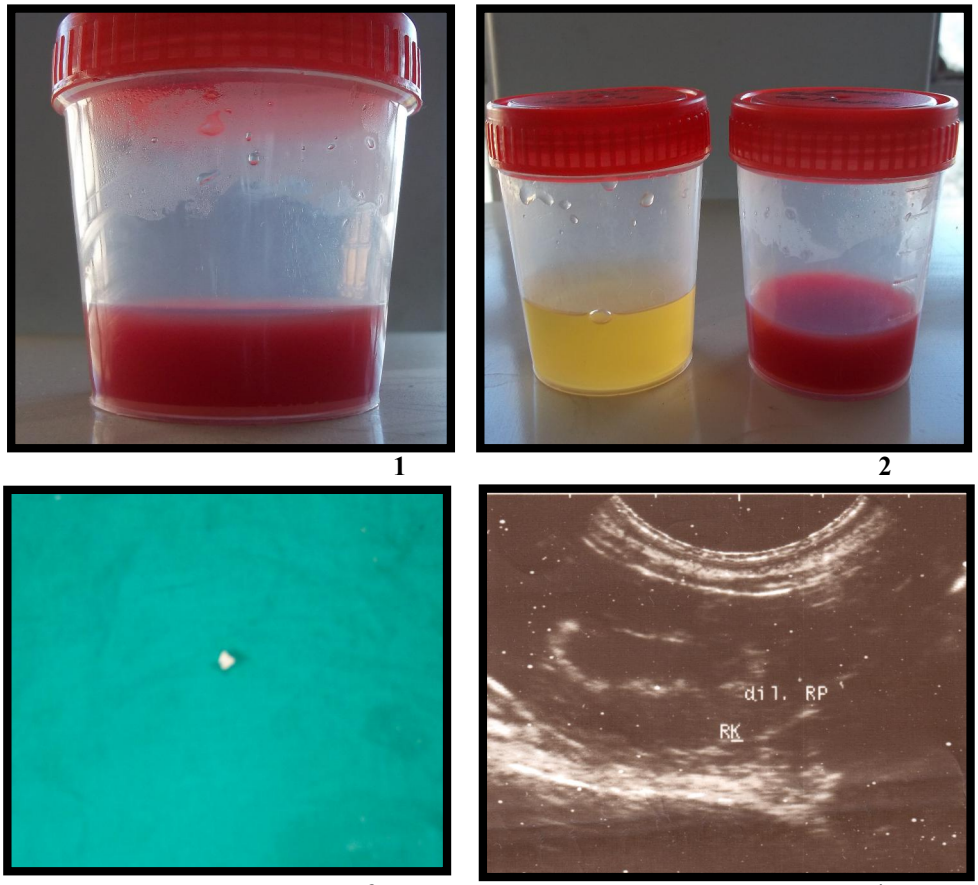
Selection of appropriate therapeutic regimen and clinical response:

Therapeutic approaches in order were either gentle manual compression of the bladder with milking of penile urethra (7 cases responded out of 42) or catheterization (27 cases responded out of 42) or cytocentesis followed by Perineal urethrostomy (8 cases responded out of 42).

Table (2): Percentage of therapeutic response of cases of FUS to different therapeutic approaches:

Therapeutic approach	Percentage (%)
gentle manual compression of the bladder with milking of penile urethra	16.7
catheterization	64.3
cytocentesis followed by Perineal urethrostomy	19

Medical treatment of the obstructive episode and in the post–obstruction period and the prevention of recurrence of blockage in the longer term were taken 12 days- 3 months (3 cases after surgical intervention).



Figures (I): Pictures of urine samples, struvite and scans of kidneys:

1-Corkscrew capped bottle contained bloody urine obtained from 3.8 Ys old Persian tomcat by catheterization. 2- 2 Corkscrew capped bottles, the first contained bloody urine (right) obtained from 7.4 Ys old Persian tomcat by catheterization and the second sample contained turbid urine (left) obtained from 4.6 Ys old Persian tomcat by gentle squeezing of urinary bladder and milking of penile urethra. 3- Struvite characterized by toothpaste like appearance obtained during catheterization of 8.5 Ys old Persian tomcat. 4-Longitudinal scan of right kidney (RK) of 4.2 Ys old Persian tomcat showed hypoechoic renal cortex with anechoic medulla and hypoechodensity of renal pelvis (dilated renal pelvis, Dil. R.P.).

4. Discussion

In male cats, a urinary tract infection can quickly progress to a life-threatening emergency. Male cats are prone to urinary blockage from lower urinary tract infections, since their urethras as longer and narrower than female cats. Since a blockage like this can become fatal in a matter of hours, a male cat showing symptoms of FUS should be taken to a veterinarian or animal hospital without hesitation. While natural treatment can be used as a complement to conventional treatments, it should not be used alone for male

All recorded cases in the present study were Persian tomcats of age between 2.7- 10 years old. Feeding of 37 out of 42 tomcats (88.1%) were commercial dry food. Seventeen tomcats were castrated (17 out of 42, 40.5%) While **Osborne et al. (1989)** mentioned that idiopathic FUS and urolithiasis are recognized in middle aged cats (2- 6 Ys of age) that are neutered**.** It is possible that urethral plugs and uroliths are caused by similar mechanisms; however, urethral plugs differ from uroliths in that they have the consistency of tooth paste and are composed of matrix. More than 90% of urethral plugs in cats are composed of struvite. Urethral plugs appear to be the most common cause of the urethral obstruction in cats **(Leib and Monroe, 1997)**. Also, **Leib and Monroe (1997)** conducted that idiopathic feline urological syndrome (FUS) probably was multifactorial. Risk factors that may predispose to development of idiopathic FUS include neutering, obesity, decreased physical activity, consumption of dry food, and decreased water intakes. In cats with naturally occurring signs of lower urinary tract disease, a cause cannot be found in 55 %. The most common urolith in cats is struvite occurring in approximately in 62 % to 88 % of cases **(Ling et al., 1990 and Osborne et al., 1992)**. In contrast to dogs, most struvite uroliths in cats are not associated with urinary tract infection; an exception is immature cats (<1 year of age), who usually develop struvite uroliths secondary to urinary tract infection **(Osborne et al., 1989 and Leib and Monroe, 1997)**

The reported clinical presentation in the present study of advanced cases were stranguria (n= 23), bloody urine (n= 23) and frequent licking of the urinary opening (n= 17), frequent attempts to urinate end with failure (n= 17), ishuria (n= 19), excessive salivation (n= 15), lethargy and vomiting (n= 13) while **Leib and Monroe (1997)** recorded the incidence of hematuria, dysuria and urethral obstruction in cats ranges from 0.5- 1% per year.

The thirteen cases presented by vomiting showed dilated renal pelvis by ultrasonography which denoted systemic reaction of uremia by retrograde urine return from urinary bladder. It was in agreement with that recorded by **Leib and Monroe (1997).** The most selected therapeutic regimen was catheterization (64.3%) as struvite toothpaste like and in most early cases could be removed by catheterization.

The present study was recommended to stop rapidly the offending dry food as reported high percentage (88.1%) of recorded emergent FUS cases. In another studies recorded by **Markwell et al. (1999), Forrester and Roudebush (2007), Buffington et al. (2006) and Stella et al. (2010)**, the proportion of cats in which clinical signs recurred was significantly lower (P = 0.04) in the canned diet group than in the dry diet group." (particularly if the patient is a male cat, because of the risk of urethral obstruction) At diet change time, a decrease in the amount (about 25%) of the old diet offered can be done each day until the change is complete which documented by **Forrester and Roudebush (2007)**. If necessary, small quantities of the cat's favorite food(s) such as meat or fish juice can be added to the new food to increase her interest in the diet. Modification of the nutrient content of diets has been suggested to play a role in the treatment of cats with FUS. Older literature related to "Feline Urological Syndrome" suggested reduction of "ash". Later studies suggested reduction of magnesium and modification of the nutrient composition of foods to create a more acidic urine. No evidence supports the use of these modifications for cats with FUS. Increasing the renal solute load of the diet using either salt(s) or protein also has been suggested, but not yet adequately tested in clinical trials in cats with FUS. The weak evidence for modification of intake of any particular nutrient modifications, and the stronger evidence for environmental enrichment (of which food certainly is a part, as described above), suggest that such modifications may not be first line treatment for cats with FUS. Cats need a high protein diet to maintain a low (acidic) urinary pH. Commercial dry cat foods are based on vegetable ingredients that make up 50% or more of the products weight, and vegetable ingredients result in the production of a high (alkaline) urinary pH. Also, the species of our domestic cat (Felis Silvestris Catus) has evolved as desert dwelling animals, capable of concentrating urine to a high degree and of relying entirely on the moisture content of prey, without the need for supplemental drinking water. Drinking is not natural for cats and they will do so only reluctantly. A cat eating 1 cup (85 g) of dry food would need to drink 8 oz. (225 ml) of water to prevent dehydration. However – most don't. The result is dehydration and a reduction in urine volume. Experiments have

shown that crystal formation of mineral ions can be prevented by increasing urine output, and the occurrence of FUS in cats fed experimental dry diets has been abolished by hydrating the same diet to a moisture content of 80%.

Pet food manufacturers literally exploit the high occurrence of disease in the modern companion cat population to market more expensive specialty diets that are supposed to address the disease cause. Numerous brands of cat foods are available that claim to prevent the occurrence of struvite crystals, most of which are – ironically – dry foods. Although the facts of how the majority of FUS cases could be prevented are clearly laid out, such commercial diets are not truly therapeutic. Pet food manufacturers are making use of a chemical substance called Ammonium chloride to artificially lower urine pH, while ignoring all dietary factors that could resolve the matter naturally. Ammonium chloride is a strong acid, and the veterinary community has since raised concern regarding its use as a means to lower urinary pH, because its acidifying affect can cause a condition called chronic acidosis. Repeated ingestion of this chemical compound, inducing acidosis, can lead to mobilization of calcium from bone – meaning that calcium is leached from the skeletal structure and deposited in soft tissue. A combination of induced acidosis, leading to calcium loss from the bones, in combination with a reduced dietary intake of magnesium leads to formation of oxalate stones – a now common side effect of the treatment for struvite crystals. Formation of oxalate stones, which are commonly found as kidney stones, but also in the heart or lower urinary tract, is the result of free calcium ions binding to oxalic acid – a substance excreted in the urine – to form the less soluble salt called calcium oxalate. Over-supplementation with calcium, dietary intake of ammonium chloride leading to calcium loss from the bones, a magnesium deficiency, supplementation of vitamin C – which is metabolized to oxalic acid – and consumption of oxalic acid containing foods – like cabbage, spinach, beet tops, potatoes, or peas – can lead to formation of calcium oxalate and the possible formation of oxalate stones in soft tissue, or the upper and lower urinary tract as described by **Robert Garrison et al. (1995), Gayla and John (1996); and James et al. (1997)**

Funaba et al. (2003), Funaba et al. (2004), Tsukada et al. (1994) Westropp et al. (2003), Dramard al. (2007) concluded that the 2 main risk factors for struvite crystals are alkaline urine and Concentrated urine. The biggest culprit for the 2 risk factors is grain-based kibble. Grains and plant matter can contribute to alkaline urine; meat on the other hand naturally promotes slightly acidic (6.0-6.5 pH) urine. Dry food typically contains 10% moisture and given cats simply cannot and will not drink enough water to make up the deficit (prey/canned food contains at least 75% moisture), they are chronically dehydrated leading to concentrated urine. Meat however with its high moisture content keeps cats' urine appropriately dilute. Long-term there is no better way to keep FUS at bay than by ditching kibble, and switching to a raw diet (or grain-free).

It was concluded that when signs of emergent FUS was presented, rapid confirmation of veterinarian by palpation of the rigid firm urinary bladder must be performed. FUS was aggravated when vomiting was presented and dilation of renal pelvis was detected by ultrasonography. Catheterization was the most selected therapeutic regimen detected by the present research workup as life saving treatment. It was recommended to adjust diet to prevent recurrence of such cases. Further studies were advised to detect the nature of the struvite and what materials dissolving struvite either by medical or alternative therapy.

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