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FELINE UROLOGIC SYNDROME (FUS). II. CHANGES IN SOME CLINICAL AND LABORATORY INDICES

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Summary

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The continuous urethral obstruction in feline urologic syndrome (FUS) results in various systemic disturbances. The present study was performed on 46 intact (uncastrated) male cats with FUS during the period 1998-2000.

The routine clinical examinations consisted in determination of body temperature, heart and respiratory rates, the presence of vomiting (nausea), anorexia, depression, dehydratation and complete urethral obstruction.

Blood samples obtained from *v. cephalica antebrachii* were analyzed for determination of haemoglobin content, haematocrit, red blood cells (RBC) and white blood cells (WBC) counts, total protein, urea, creatinine, calcium and phosphate concentrations.

The results of all those studies showed that the continuous urethral obstruction (more than 24 hours) resulted in rapidly progressing uraemia with significantly increased blood urea and creatinine, signs of anorexia, vomiting, dehydratation, bradycardia or tachycardia, polypnea, hypothermia. When blood urea levels were raised over 50 mmol/l an uraemic comma and lethal issue could be anticipated, so it was determined as a critical threshold. In FUS, the signs of lower urinary tract inflammation usually prevailed whereas the involvement of the proximal urinary tract was relatively rarely observed.

Key words: feline urologic syndrome, FUS, uraemia, urolithiasis

INTRODUCTION

The term feline urologic syndrome (FUS) was initially introduced by Osbaldiston and Taussig (1970) to designate a complex of symptoms, clinically manifested by the disuria, haematuria and urethral obstruction triad (Willeberg, 1981; Barsanti et al., 1982).

The problems related to FUS consist mainly in precise diagnostics and evaluation of the clinical status in order to the timely choice of a correct solution for intervention, that in most cases is perineal urethrostomy (Aminkov et al.,1999).

There are significant differences in the clinical picture of the disease related to the gender. The incidence of the non-obstructive form is equal in male and female cats while the obstructive form affects particularly male individuals (Senior, 1999). This is related to the particularities of urethral anatomy in male cats. Male feline urethra is divided into four segments – preprostatic, prostatic, postprostatic and penile parts with progres-

sively diminishing diameter towards the issue (Christie, 1985). This anatomy is a prerequisite for frequent obstructions caused by urethral plugs. The condition of obstructed patients is critical and requires a rapid diagnostics and urgent conservative and/or operative treatment (Labato, 1992).

The aim of the present study was to follow out the deviations in some clinical and laboratory indices in this disease and to evaluate the severity of occurring uraemia with regard to the precise choice of a therapeutical approach.

MATERIALS AND METHODS

The study was performed on 46 male intact (uncastrated) cats with signs of disurua, haematuria and urethral obstruction persisting for more than 24 hours, admitted for diagnostics and treatment to the Surgical Clinic of the Faculty of Veterinary Medicine, Trakia University for a 2-year period (1998-2000).

The information for the clinical status included the body temperature (^{o}C), heart and respiratory rates (min^{-1}), presence of appetite, presence of urethral obstruction (established by catheterization), vomiting, dehydratation.

Blood samples were obtained from *v. cephalica antebrachii* for determination of the following laboratory indices:

A. *Haematological*: haemoglobin (colorimetrically, g/l); haematocrit (by microcentrifugation, l/l), red blood cell (RBC, T/l) and total white blood cell (WBC, G/l) counts - by counting in a Bürker chamber;
B. Biochemical: total protein (g/l); urea (mmol/l), creatinine (μmol/l), calcium (mmol/l) and inorganic phosphate (mmol/l) – with commercial kits of Human Diagnostica, Germany.

RESULTS

The clinical examination of studied patients showed that more than half of all affected animals (53.1%) were with a normal body temperature, in 47.7% it was subnormal and only in 0.2% - increased. The heart rate was accelerated in 64.2% of cats, slowed in 7.9% and normal – in 27.9%. The respiratory rate was higher than the upper reference range limit in most patients (78.6%) while in others no deviations were observed (Table 1). Other clinical signs as anorexia (60%), vomiting (44.2%), dehydratation (55.6%) and complete obstruction (78.3%) were also present (Table 2).

The results from the haematological study showed no changes in RBC counts, blood total protein and total calcium concentrations (Table 3). A haemoconcentration, evidenced by the increased haema-

Table 1. Clinical parameters in FUS patients; mean ± SEM (n=46)

Parameter	Value	Percentage*
Body temperature, ^{o}C	37.8 ± 0.17	↓ 46.8 ↑ 0.2
Heart rate, min^{-1}	140.8 ± 5.95	↑ 64.2
Respiratory rate, min^{-1}	43.10 ± 2.94	↑ 78.6

↑ - higher than normal; ↓ - lower than normal; * cases with deviations to the total number.

tocrit and haemoglobin levels (in 21.7% and 35.7% of all patients respectively) was present.

Table 2. Incidence of characteristic clinical signs in FUS patients (n=46)

Parameter	Presence (%)	Absence (%)
Anorexia	60.0	40.0
Vomiting	44.2	55.8
Urethral obstruction	78.3	21.7
Decreased skin elasticity	55.6	44.4

Table 3. Laboratory parameters in FUS patients; mean ± SEM (n=46)

Parameter	Value	Percentage*
Haemoglobin, g/l	143±5	↑ 35.7
Haematocrit, l/l	0.42±0.01	↑ 21.7
RBC, T/l	7.6±0.2	-
WBC, G/l	14.2±1.3	↑23.3
Total protein, g/l	63±2.3	-
Urea, mmol/l	26.4±4.0	↑ 58.0
Creatinine, μmol/l	650±232	↑ 66.6
Calcium,mmol/l	2.29±0.06	-
Phosphate, mmol/l	1.63±0.11	↑ 33.3

↑ - higher than normal; ↓ - lower than normal; * cases with deviations to the total number.

A leukocytosis was observed in 23.3% of cats while in the other 76.7% no pathological changes in WBC counts existed. The changes in urea and creatinine levels were the most significant. Those parameters were increased respectively in 58% and 66.6% of patients. The levels of blood inorganic phosphate were elevated in 33.3% of all cats.

DISCUSSION

The obstructive diseases of the lower urinary tract in male cats are life-threatening because of the rapidly developing uraemia –within 24-72 hours (Labato, 1992), what is categorically evidenced by the results of both clinical and laboratory examinations.

Many FUS researchers (Osborne et al., 1995a; Osborne et al., 1999; Senior, 1999) reported its recurrent character and acute course. According to studies of ours, the issue of the disease is directly related to its duration (Dinev et al., 2001). Hause (1984) and Senior (1999) showed that the continuous urethral obstruction caused a postrenal azotaemia, dehydratation, metabolic acidosis and hyperkaliaemia leading to lethal issue. In persistent obstruction, death occurred 3-5 days after its onset (Lage, 1998).

FUS is accompanied by a characteristic clinical picture and laboratory findings. The clinical manifestation of the syndrome is related to uraemia-mediated lesions in various organs and systems. The results of our studies evidenced that a significant percentage of the patients showed signs as subnormal body temperature, tachycardia, polypnea, vomiting, dehydratation and increased urea and creatinine concentrations. Our findings correspond to the data communicated by Labato (1992) and Senior (1999).

The total protein concentrations, being within the normal range, evidenced that the inflammation affected only the lower urinary tract. A confirmation of this are the results of urine sediment analysis in FUS patients, reported in a previous study of ours (Dinev et al., 2001). They showed signs of lower urinary tract inflammation: prevalence of epithelial cells originating from the bladder, limited leukocyturia, weak proteinuria and significant haematuria. The leukocytosis (in 23.3% of cats) and the presence of epithelial cells from the renal pelvis in urine (Dinev et al., 2001) could be accepted as an evidence

for a certain permeation of infection in kidneys as well.

According to Osborne (1995b) the hypercalciaemia is not a characteristic or accompanying sign in calcium oxalate urolithiasis. Plasma calcium concentrations in the cats included in the study were not elevated while those of inorganic phosphate were over the upper reference range in 33.3% of cases with struvite urolithiasis. This could be related to the increased ingestion of phosphorus with food (Dinev et al., 2001). A hyperphosphataemia is also present when phosphates are retained in blood because of a decreased glomerular filtration rate but this is more characteristic for the chronic renal failure (Barber and Elliott, 1998).

The levels of blood urea and creatinine in our study were above the physiological values. Both parameters are with an important diagnostical value in some renal disturbances and their deviations are indicative for azotaemia, probably because of urine retention and respectively, the retention of toxic metabolites (Hall, 1987). It could be accepted that there are prerequisites for toxic damage of the renal parenchyme and symptoms of acute renal failure.

Hause (1984) and Labato (1992) reported that the continuous urinary obstruction is accopmanied by a severe metabolic acidosis developing as result of enhanced catabolism following the intoxication. Thus, the intracellular water and electrolyte exchange is impaired and manifestations of hyponatriaemia, hyperkaliaemia and hypertonic dehydratation occur (Morgan, 1983; Chew and DiBartola, 1986). The dehydration was further confirmed by the decreased skin elasticity, the increased haemoglobin and haematocrit values – symptoms, observed in some animals included in the study. Those changes reflect upon the cardiovasculary, alimentary and cerebral activities and result in bradycardia, followed by tachycardia and hypertony, polypnea, vomiting, anorexia, hypothermia, lethargy and comma. Each of these signs was manifested at a different extent during the course of the disease in our patients. The respiratory rate was accelerated, probably as a response of oxygen insufficiency caused by metabolic acidosis. The irritation of the gastric epithelium by nitrogen compounds likely leads to vomiting and anorexia. The depression and hypothermia could be explained by the toxic damage and hypoxia of brain with resulting brain oedema as a non-specific reaction to oxygen insufficiency (Stefanov, 1983).

According to Landes et al. (1984), FUS is one of the principal causes for death in the feline species (7%) together with panleukopenia (16.6%), traumatism (11.5%), feline infectious peritonitis (6%), circulatory (5.7%) and alimentary (5.3%) disorders.

In two cases, the issue was lethal despite the corrective fluid and electrolyte (sodium hydrogen carbonate and calcium borogluconate) therapy. In those patients blood urea levels were higher than 50 mmol/l, giving grounds to consider that this could be the critical threshold for development of uraemic comma and death. A particular attention should be paid on the impaired sodium reabsorption in renal tubules due to the acute renal failure. The higher sodium concentration in the distal tubule stimulates renin secretion, respectively angiotensin II, resulting in afferent arterioles spasm and renal ischaemia (Stefanov, 1983; Chew and DiBartola, 1989). It is known that renal structures are very sensitive to ischaemia and in such conditions, the ultrastructural changes in the tubular epithelium occur as early as in few

hours and they are later accompanied by cytoplasm vaccuolization and tubular necrosis. That is why the correct treatment should be urgently initiated. The therapeutical approach could be bidirectional – either conservative or operative. The conservative treatment is directly related to the etiology (Hause, 1984). The operative technique solving radically the problem, is the perineal urethrostomy according to Wilson and Harrison.

CONCLUSIONS

The continuous urinary obstruction (more than 24 hours) resulted in rapidly progressing uraemia with increased blood urea and creatinine levels, signs of anorexia, vomiting, dehydratation, bradycardia or tachycardia, polypnea, hypothermia.

When blood urea levels were raised over 50 mmol/l. an uraemic comma and lethal issue could be anticipated, so it was determined as a critical threshold.

The predominating signs in FUS are those of lower urinary tract inflammation. The proximal urinary tract was less commonly affected.

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