Emphysematous cystitis in a non-diabetic cat with *Staphylococcus* spp.

Lora Koenhemsi1*, Belgi Diren Sigirci2, Remzi Gönül1, Erman Or1

1. Istanbul University-Cerrahpasa, Veterinary Faculty, Department Internal Medicine, Avcilar, 34320, Istanbul/Turkey.
2. Istanbul University-Cerrahpaşa, Faculty of Veterinary Medicine, Department of Microbiology, Avcilar, 34320, Istanbul/Turkey.


ABSTRACT

Emphysematous cystitis (EC) is uncommon infection of urinary tract which is characterized by air within the bladder wall and lumen. A 3 years old male cat presented with polyuria/polydipsia, lethargy, and inappropriate urination. Temperature, pulse and respiratory rates were within normal limits on physical examination. Blood work revealed leukocytosis and increased levels of blood urea nitrogen and creatinine. Urinalysis revealed persistent proteinuria in urine dipstick and bacteria under microscopical examination of urine. The presence of air in the bladder were seen in latero-lateral abdominal view. Based on lab work and imaging abnormalities, a diagnosis of emphysematous cystitis was made. The use of diagnostic imaging and lab work are necessary in the diagnosis and treatment of this rare disease, and will be described in this report.

Keywords: Emphysematous cystitis, cat, *Staphylococcus* spp, radiology

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Introduction

Emphysematous cystitis (EC) is uncommon bacterial infection of urinary tract which is characterized by air within the bladder wall and lumen. EC cases have been occurred in humans, dogs, cats and a cow (Moon et al., 2014). EC has reported in dogs more than cats. Although no sex predilection has been reported in animals, female patients more likely to be affected by EC in dogs (Moon et al., 2014; Petite et al., 2006).

Etiology is multifactorial and pathogenesis is poorly understood. These infections are often reported with Diabetes mellitus in humans, and animals. Slightly more than half of the cases (15 of 26) had underlying diabetes mellitus in dogs and cats (Moon et al., 2014). Also EC has been diagnosed with primary renal glycosuria (Fanconi’s syndrome), urinary tract obstruction, chronic urinary tract infections, neurogenic bladder dysfunction, morphologic abnormalities, iatrogenic (e.g., cystocentesis, catheterization, cystoscopy) and immunosuppression (Moon et al., 2014). Early recognition of EC is important in order to prevent progression of the infection to emphysematous pyelonephritis and urosepsis (Fabbi et al., 2016).
A 3 years old male cat presented with polyuria, polydipsia, lethargy, and inappropriate urination. On the initial physical examination, the patient was dehydrated. Temperature, pulse and respiratory rates were within normal limits.

Initial diagnostics included a complete blood count, serum biochemical profile, urinalysis, and abdominal radiographs. Blood work revealed leukocytosis and increased levels of blood urea nitrogen and creatinine. Glucose was within normal levels (Table 1). Urinalysis, obtained by cystocentesis, and revealed proteinuria (3+) and hemoglobinuria (3+) in urine dipstick. A large amount of erythrocytes and bacteria, 8-10 leukocytes/high-power field (hpf), 3-5 epithelial cells/hpf and 1-2 struvite crystals/hpf were detected in the microscopic examination. A urine sample was submitted for culture and antibiotic sensitivity.

In abdominal radiograph a huge amount of gas detected in the middle of the urinary bladder lumen (Figure 1a). An iodine-based, water soluble contrast media was given to the cat by urinary catheter which can be seen at the caudo-dorsal of the bladder. Multiple round gas opacities within contrast media in the bladder and urethra was detected (Figure 1b). Based on lab work and imaging abnormalities, a diagnosis of emphysematous cystitis (EC) was made. Initial treatment started with Enrofloxacin (5-10 mg/kg, IM BID) and bladder drainage.

Urine culture was positive for Staphylococcus spp, and found resistant to Ampicillin, Amoxicillin/clavunolic acid, Eflperazon, Ciprofloxacin, Cefovecin, Gentamicin, Marbofloxacin. Sensitive to Oxytetracycline, Enrofloxacin, and Streptomycin. According to these results initial treatment continued. After 10 days of the treatment, the patients’ complications resolved and after 20 days there was a little gas in the bladder on x-rays (Figure 2a). 40 days after the diagnosis, a routine blood analysis, urinary test and radiography were repeated (Table 1). Leukocyte count, blood urea and creatinine levels were decreased and there was a resolution of the gas in the x-rays (Figure 2b).

**Table 1:** Routine blood tests on the day of presentation and after 40 days of treatment

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Values</th>
<th>Before Treatment</th>
<th>After Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC (1x 10^6 µL)</td>
<td>6-10</td>
<td>8.3</td>
<td>6.8</td>
</tr>
<tr>
<td>HGB (g/dl)</td>
<td>9.5-15</td>
<td>11.4</td>
<td>9.2</td>
</tr>
<tr>
<td>HCT (%)</td>
<td>29-45</td>
<td>34</td>
<td>30</td>
</tr>
<tr>
<td>WBC (x10^9/µL)</td>
<td>6-19.5</td>
<td>21</td>
<td>10.1</td>
</tr>
<tr>
<td>PLT (1x10^3/µL)</td>
<td>150-600</td>
<td>55</td>
<td>159</td>
</tr>
<tr>
<td>MCV</td>
<td>41-54</td>
<td>41</td>
<td>44</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>13-17.5</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>MCHC (%)</td>
<td>31-36</td>
<td>34</td>
<td>31</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>28-76</td>
<td>54</td>
<td>48</td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>5-55</td>
<td>80</td>
<td>21</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>15-34</td>
<td>324</td>
<td>24</td>
</tr>
<tr>
<td>CRE (mg/dL)</td>
<td>0.8-2.3</td>
<td>10.4</td>
<td>1</td>
</tr>
<tr>
<td>GLU (mg/dL)</td>
<td>70-150</td>
<td>103</td>
<td>119</td>
</tr>
</tbody>
</table>

CRE = Creatinin, GLU = Glucose

Initial diagnostics included a complete blood count, serum biochemical profile, urinalysis, and abdominal radiographs. Blood work revealed leukocytosis and increased levels of blood urea nitrogen and creatinine. Glucose was within normal levels (Table 1). Urinalysis, obtained by cystocentesis, and revealed proteinuria (3+) and hemoglobinuria (3+)

**Figure 1:** a- abdominal laterolateral view of the cat, b- positive contrast abdominal laterolateral view with an iodine-based, water soluble contrast media.
The pathogenesis is not completely understood in EC cases, it has been suggested that the high tissue glucose levels and immunodeficiency can lead to infection (Moon et al., 2014). It is caused by fermentation of an underlying bacterial infection mostly by *Escherichia coli* (60%) and *Klebsiella pneumoniae* (10-20%). Other microorganisms isolated in human EC cases include *Aerobacter, Enterobacter, Proteus, Citrobacter, Staphylococcus, Streptococcus, Nocardia, Clostridium and Candida* but these cases are rare (Amano and Shimizu, 2014; Quint et al., 1992). *Clostridium* spp., *Escherichia coli*, *Klebsiella* spp., *Proteus* spp., *Aerobacter aerogenes* have been isolated in dogs and cats (Moon et al., 2014; Lobetti and Goldin, 1998). Petite et al. (2006), reviewed the medical records of dogs with ultrasonographic diagnosis of EC and isolated *Proteus mirabilis* in all dogs. This case exhibits *Staphylococcus* spp. which is rare isolated bacteria in EC cases.

It has been suggested that high glucose consumption by the microorganisms produce carbon dioxide and hydrogen thorough the natural fermentation process. In non-diabetic patients increased levels of urinary albumin, lactose, or tissue proteins can result in gas formation. Also bacterial endotoxin release contribute to the process (Bos et al., 2014; Fabbi et al., 2016).

The clinical signs of EC disease are often characterized by non-specific and very variable clinical symptoms, with little or no diagnostic clues. The most common ones are abdominal pain and hematuria which was not detected in our case (Moon et al., 2014; Amano and Shimizu, 2014). However other symptoms like dysuria and urinary frequency occur in 50% of human patients and compatible with the cat symptoms (Amano and Shimizu, 2014).

The most common imaging method for obtaining a definitive diagnosis is abdominal radiography (Amano and Shimizu, 2014). However the sensitivity is low because it is sometimes difficult to distinguish from intestinal gases (Fabbi et al., 2016). Although radiographic classification of EC has not been established for animals, we used the human classification system in our case. According to this; grade 1 is characterized by gas in the urinary bladder wall. It is classified as grade 2, when there is a thickening and irregularity in the wall. Gas both in the bladder lumen and wall is classified as grade 3 (Moon et al., 2014; Petite et al., 2006). The cat presented in this case classified as grade 3.

Ultrasonography can be useful for detection of early cases. Ultrasonographic findings include a hyperechoic stripe with reverberation artifact (Moon et al., 2014; Petite et al., 2006). However a large amount of gas has been identified in the bladder on radiographs taken at initial presentation in our case so it not useful to make an ultrasonography. Also positive contrast cystogram was performed to the cat and multiple gas opacities was detected in the urethra via this method. Maybe it'll be useful in EC cases.

Management of this disease depends on it severity and mostly consists of antibiotics, bladder drainage and glycemic control (Petite et al., 2006). The prognosis of EC is good when appropriate antibiotic therapy is given (Fabbi et al., 2016). Duration of treatment is not well established (Petite et al., 2006). Grupper et al. (2007) found that a median length of 10 days for treatment is effective. Although after 10 days there was a resolution of the complications, the gas in the urinary bladder resolved after 40 days in our case. No significant clinical signs strongly suggestive of EC have been reported to the authors knowledge; hence EC can easily be missed. A delayed diagnosis can result an over helming infection of the upper urinary tract and develop emphysematous pyelonephritis that difficult to treat and able to cause death (Amano and Shimizu, 2014). More awareness of the condition is important for the future diagnosis and treatment.

**Discussion**

Figure 2: a- abdominal view after 20 days of the treatment, arrow: small amount of gas in the bladder. b-after 40 days of the treatment.
References


