Induced Acute Renal Failure (Injury) by Administration of Ethylene Glycol (Antifreeze) in Cats

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ABSTRACT

Ethylene glycol (EG) poisoning is highly dangerous in cats and a small dose of it could cause signs which are initially non-specific and may be easily missed. So, our study was preformed to determine the various effects of EG administration on cats. 10 cats of both sexes aging 1-3 years old were included in this experimental study. Cats were examined before starting the experiment clinically, physically and ultrasonographically to ensure their soundness, then EG was administrated at a dose of 1.4 ml/kg b.wt. orally by using of sterile syringe. Clinical signs started with in 12 hours after adminstraion and 4 cases were died in the 2nd day after ingestion and the remaining 6 cats were followed up to 5th day of EG administration. Clinical, physical, , urine, ultrasonographic and post-mortem examinations were preformed on each cat before EG ingestion and on the 2nd and 5th day after EG ingestion. In addition, Blood samples were collected from each cat for hematological and serum biochemical analysis. Our results revealed that, clinically the cats were depressed, anorexic, unwilling to move with decreased rectal temperature and pulse rate while the respiratory rate was significantly increased at the 5th day of EG ingestion. Hematological analysis showed a significant increase of erythrocytic count while hemoglobin and PCV levels were significantly decreased at the 5th day of the experiment. The total leucocytic count was significantly increased with significant neutrophilia and lymphocytopenia at the end of the experiment. Serum analysis of these cats showed azotemia, reduction in calcium, potassium and chloride with elevated phosphorus, sodium and magnesium levels. Urinalysis showed calcium oxalate crystalluria which could be noticed shortly after EG ingestion. It could be concuded that ethylene glycol ingestion could cause a sever renal damage in cats which may be fatal and methods of prevention of its ingestion by cats should be applied to avoid its serious effects.

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1. INTRODUCTION

Ethylene glycol (antifreeze) poisoning is one of the most life-threatening toxins that observed in pets (dogs and cats). It is a major component of many commercial antifreeze products and it is considered as a common etiological factor causing acute renal failure in dogs and cats (Connally et al., 2010). Cats are very sensitive to the effects of ethylene glycol and even a small quantity is hazardous. The lethal dose of ethylene glycol is very low in cat (1.5 ml/kg b.wt) as mentioned by Beasley and Buck, 1980. The morality rate is seriously high as it ranged from 96-100% in cat (Rowland, 1987). Cat could ingest it because of palatable taste and warming sensation and once it ingested, the clinical signs could be developed within 30 min. the injuries mainly affecting the real parenchyma with development of acute renal failure. Liver could be also affected and sever toxic hepatitis could develop. The narcotic effect of noxious substance, its metabolites or endotoxic products at later stage would cause cerebral damage. Sever toxic hepatitis could be also developed (Goicoa et al., 2003).
In case of acute ethylene glycol intoxication, pulmonary edema, uremic pneumonia and hyperemia of the gastric mucosa could be also observed (Marcato, 2002). Thrall et al., 1995 divided the clinical signs of ethylene glycol intoxication into 3 phases:

Phase I: occurs 30 min.-12 hours after ingestion and signs are similar to alcohol intoxication including polyuria, vomiting, depressed mentation, ataxia, nausea and polydipsia.

Phase II: occurs 12-24 hours after ingestion and it characterized clinically by rapid or distressed respiration.

Phase III: usually observed at 24-72 hours after ingestion of ethylene glycol and owners may observe diarrhea, nausea, vomiting, increased or decreased urination, anorexia and severe depression. These signs are associated with acute renal failure.

Urinalysis is a critical diagnostic tool in the evaluation of suspected ethylene glycol intoxication. Urinalysis will often be minimally concentrated and reveal calcium oxalate crystals in the urine. As the degree of toxicity progresses, other changes such as blood, protein, glucose, and inflammatory cells may be seen in the urine (Osweiller, 1996).

Hemogram of affected animals may show neutrophilia and lymphopenia with increased packed cell volume (Beasley and Buck, 1980). Serum biochemical analysis may show azotemia (elevated urea and creatinine), hypophosphatemia, hypocalcemia, hyperglycemia, hyperkalemia, and sodium concentration was usually normal (Osweiller, 1996).

The ultrasonographic changes varied from mild to marked increase in renal cortical echogenicity a pattern of greater than normal cortical and medullary echogenicity with persistence of areas of lesser echointensity at the corticomedullary junction and central medullary regions was observed. This pattern, termed the halo sign. (Adams, et al., 1991).

Aim of work: This study was preformed to detect the nephrotoxic effect of ethylene glycol (antifreeze) ingestion on cat regarding to the clinical signs, the hematopoietic alterations in blood of the affected cats and the ultrasonographic imaging of the kidneys of cats suffered from ethylene glycol intoxication. Furthermore, to reveal the usefulness of ultrasonography, postmortem examination and urinalysis as confirmatory tools in diagnosis of such renal affections that caused by antifreeze ingestion in cat.

2. MATERIAL AND METHODS:

2.1. Animals:

This study was performed on 10 cats (6 males and 4 females) aging 1-3 years old. These animals were examined clinically and ultrasonographically before starting the experiment to ensure their soundness. Then, food was withheld from all animals under experiment for 8 hours prior to starting the experiment. After that all animals were administrated ethylene glycol by a dose of 1.4 ml/kg body weight orally by using of sterile syringe.

2.2 Physical examination:

Rectal temperature, pulse rate and respiration rate of each animal were measured before administration of ethylene glycol and at 2nd and 5th day post-administration.

2.3. Sampling:

2.3.a. Urine samples:

It was collected from each animal before administration of ethylene glycol and at 2 and 5 days post-administration, using tom cat catheter130mm manufactured by value line cat catheter×1.3 into clean sterile bottle secured to the urethral catheter introduced into animal to collect a fresh urine sample then send into a lab in a maximum one hour time to perform complete urine analysis.

2.3.b. Blood samples:

Two Blood samples were taken under sedation with (Xylazine HCl, 1mg/kg B.W. I/M) by puncture of cephalic vein before administration of ethylene glycol and at 2nd and 5th day post-administration.

2.3.b.1. First blood sample (coagulated blood):

Blood samples were collected in test tubes without anticoagulant, left at room temperature for 1 hour and kept in the refrigerator for another two hours then centrifuged at 1500 rpm for 10 minutes, then transferred serum to sterile clean vials and stored at (-20 C) for biochemical analysis for serum urea nitrogen (kits produced by Bioscope diagnostic), creatinine (kits produced by Bioscope diagnostic), total protein (kits produced by biodiagnostic diamond), calcium (kits produced by biodiagnostic diamond), inorganic phosphorus (kits produced by bioscope diagnostic), magnesium and chloride (kits produced by bycentronic GmpH), and sodium & potassium (kits produced by Greiner).
2.3.b.2. Second blood sample (non coagulated blood):
Blood samples were collected into sterilized test tubes containing EDTA as anticoagulant then genital inversion of the tubes for several times for hematological examination including RBCs count, hemoglobin, PCV, total and differential leucocytic count.

2.4. Urinalysis:
Urine samples were examined physically, chemically, macroscopically and microscopically at the same days mentioned before as method described by Kelly (1984)

2.5. Ultrasonographic examination:
After sampling, animals were examined ultrasonographically by using Mindray ultrasound for determination of different affections before administration of ethylene glycol and at 2nd and 5th day post-administration.

2.6. Postmortem lesions: kidneys were collected from each animal directly after death at day 2 and day 5 from EG ingestion.

2.7. Statistical Analysis: All variables were reported as mean ± standard error of mean. A probability value ≤ 0.05 was considered statistically significant. All analyses were performed using the SPSS statistical software package (version 22.0 SPSS Inc., Chicago, USA). Tests of normality of data and homogeneity of variances were performed on raw data. Positively skewed variables were log-transformed (with back-transformation for reporting) and re-tested for normality and homogeneity. For normally distributed data, one-way analysis of variance (ANOVA) with the stage of experiment as the main effect was performed followed by Tukey-Kramer test for multiple comparisons. For unequal variance, Welch’s F-test and Games-Howell post-hoc test were used.

3. RESULTS:
3.1. Clinical findings:
Four cats were died within 12-36 hours of EG ingestion after suffering from nervous signs either depression or convulsions while the survived cats (6 cats) were suffered from anorexia and depression at 2nd day of EG ingestion, then at the 5th day they suffered from Ataxia, vomiting, anoxia, pale gums, severe dehydration, depression, loss of coordination, decreased urine production, the body temperature, respiration rate and the pulse rate were significantly decreased after EG ingestion as compared with their levels before starting the experiment, as described in table (1).

3.2. Serum and hematological analysis:
a. Serum analysis of cats under the experiment showed that, the BUN and creatinine levels were significantly increased after EG administration as compared with their levels before administration. While serum total protein, was significantly decreased post-administration than before EG administration, (table 2)

Regarding to serum calcium level in examined cats, it was significantly decreased at 5th day of EG administration than its level before administration. Serum phosphorus and sodium levels were significantly increased post administration that before EG administration.

The serum chloride and potassium levels, were significantly decreased at 5th day of EG administration than their levels before administration, while serum magnesium level was significantly increased at 5th day after EG administration than its level before starting of the experiment. (table 2).

b. Hematological examination of blood of animals under the experiment revealed that, RBCs count was significantly increase after administration of EG as compared with its level before administration, while haemoglobin concentration and PCV %, were significantly increased in post-administration of EG than before starting of the experiment. (table 3)

Table(1): Effect of ethylene glycol ingestion on rectal temp., respiratory rate and pulse rate of cats:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before administration of ethylene glycol</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectal temp.,C⁰</td>
<td>38.9±0.14⁴</td>
<td>36.0±0.37⁵</td>
<td>35.9±0.19⁶</td>
</tr>
<tr>
<td>Pulse rate (beat/min)</td>
<td>152±5.12⁴</td>
<td>174±2.08⁵</td>
<td>180±2.24⁴</td>
</tr>
<tr>
<td>Respiratory rate (/min)</td>
<td>34.7±1.33⁵</td>
<td>41.2±1.19⁶</td>
<td>43.8±1.3⁴</td>
</tr>
</tbody>
</table>

Values are means ± standard errors.
Means without a common superscript are significantly (P<0.05) different.
Regarding to the WBCs count, blood plateles,neutrophils and lymphocytes, there was a significant increase in their levels after EG administration as compared with their levels before starting of the experiment.(table 4).

3.3. Urinalysis:

The results of urinalysis of cats under experiment revealed that physically, there was hematuria in all cats administrated EG with increased specific gravity and acidic PH, while chemically, oxalate crystals can be noted after ingestion of EG in all cats.

Table (2): Effect of ethylene glycol on some serum biochemical parameters in cat:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before administration</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood urea nitrogen, mg/dl</td>
<td>14.1 ± 0.14&lt;sup&gt;a&lt;/sup&gt;</td>
<td>63.1 ± 12.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>257.0 ± 5.14&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td>1.56 ± 0.27&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10.71 ± 1.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>21.83 ± 1.53&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total protein, g/dl</td>
<td>7.90 ± 0.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.53 ± 0.03&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.60 ± 0.08&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Calcium, mg/dl</td>
<td>9.95 ± 0.26&lt;sup&gt;c&lt;/sup&gt;</td>
<td>9.35 ± 0.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.85 ± 0.16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Phosphorus, mg/dl</td>
<td>7.23 ± 0.33&lt;sup&gt;c&lt;/sup&gt;</td>
<td>9.54 ± 0.07&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10.74 ± 0.08&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium, mEq/l</td>
<td>153.8 ± 1.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>163.5 ± 0.91&lt;sup&gt;b&lt;/sup&gt;</td>
<td>169.8 ± 1.82&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Chloride, mEq/l</td>
<td>124.2 ± 1.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>115.5 ± 0.26&lt;sup&gt;b&lt;/sup&gt;</td>
<td>115.1 ± 0.18&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Potassium, mEq/l</td>
<td>4.54 ± 0.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.72 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.63 ± 0.05&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Magnesium, mEq/l</td>
<td>1.97 ± 0.05&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.08 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.48 ± 0.16&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± standard errors. Means without a common superscript are significantly (P<0.05) different.

Table (3): Effect of ethylene glycol on hematological parameters in cat:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before administration</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBCs, 10&lt;sup&gt;6&lt;/sup&gt;/ml</td>
<td>4.57 ± 0.14&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.56 ± 0.26&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.47 ± 0.4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hemoglobin, g/dl</td>
<td>12.0 ± 0.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.5 ± 0.23&lt;sup&gt;c&lt;/sup&gt;</td>
<td>11.2 ± 0.16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Packed cell volume, %</td>
<td>40.1 ± 0.46&lt;sup&gt;c&lt;/sup&gt;</td>
<td>41.53 ± 0.62&lt;sup&gt;b&lt;/sup&gt;</td>
<td>43.63 ± 0.38&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± standard errors. Means without a common superscript are significantly (P<0.05) different.

Table (4): Effect of ethylene glycol on total and differential leucocytic count:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before administration</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC, 10&lt;sup&gt;3&lt;/sup&gt;/ml</td>
<td>11.6 ± 0.47&lt;sup&gt;c&lt;/sup&gt;</td>
<td>33.0 ± 4.87&lt;sup&gt;b&lt;/sup&gt;</td>
<td>59.3 ± 11.2&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Blood platelets count, 10&lt;sup&gt;3&lt;/sup&gt;/ml</td>
<td>210.9 ± 8.81&lt;sup&gt;b&lt;/sup&gt;</td>
<td>233.5 ± 12.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>312.0 ± 35.4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Blood neutrophils, %</td>
<td>48.2 ± 2.23&lt;sup&gt;b&lt;/sup&gt;</td>
<td>85.2 ± 2.52&lt;sup&gt;a&lt;/sup&gt;</td>
<td>90.8 ± 1.01&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Blood lymphocytes, %</td>
<td>43.1 ± 2.18&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.20 ± 0.53&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.00 ± 0.68&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± standard errors. Means without a common superscript are significantly (P<0.05) different.

Table (5): Effect of ethylene glycol on some urine parameters:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before administration</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity</td>
<td>1.018 ± 0.001&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.028 ± 0.002&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.037 ± 0.003&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pus cells</td>
<td>2.1 ± 0.23&lt;sup&gt;b&lt;/sup&gt;</td>
<td>14.0 ± 2.83&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.0 ± 1.44&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>RBCs</td>
<td>2.19 ± 0.11&lt;sup&gt;c&lt;/sup&gt;</td>
<td>28.2 ± 7.89&lt;sup&gt;b&lt;/sup&gt;</td>
<td>331.1 ± 29.8&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are means ± standard errors. Means without a common superscript are significantly (P<0.05) different.
Table (6): Chemical changes of urine after administration of ethylene glycol:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before administration</th>
<th>2 days post-administration</th>
<th>5 days post-administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Nil</td>
<td>Few</td>
<td>&gt;500</td>
</tr>
<tr>
<td>Urobilinogen</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Bilirbin</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Protein</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Nitrite</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Ketones</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Glucose</td>
<td>Nil</td>
<td>+ve</td>
<td>+ve</td>
</tr>
<tr>
<td>PH</td>
<td>6.5</td>
<td>7.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1020</td>
<td>1030</td>
<td>1035</td>
</tr>
<tr>
<td>Leucocytes</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>

3.4. Ultrasound examination of cats administrated EG:

Our obtained results of the kidneys showed that, a diffuse hyperechogenic cortical with small shadow cones, suggestive for nephrocalcinosis.

Fig. (1) Ultrasoundography of normal kidney before administration of ethylene glycol show hypoechoic renal cortex (C) than liver (L).

Fig. (2): Ultrasoundography of kidney 2 day after ingestion of nephrotoxic agent. Renal cortex (C) still hypoechoic than liver (L).

Fig. (3): Ultrasoundography of kidney 5 days after toxin ingestion, Renal cortex (C) hypoechoic than liver (L).

Fig. (4): Ultrasoundography of R. kidney 5 days after toxin ingestion show Diffuse renal calcinosis. Hyperechogenic cortical and medullar.
4. **Post-mortem findings:**

Our obtained results showed that, the kidneys were pale, globular, wrinkled surface and showed discrete cortical petechiae. The longitudinal section of right kidney showed dilated renal pelvis and filled with urine with parenchymal atrophy due to over pressure by excess fluid, while the left kidney was atrophied (fig.6-10).

![Ultrasonography of left kidney showing renal calculi and crystal mineralization of the kidney.](image)

**Fig. (5):** Ultrasonography of left kidney showing renal calculi and crystal mineralization of the kidney.

![The kidneys were pale, globular, wrinkled surface and showed discrete cortical petechiae](image)

**Fig. (6):** The kidneys were pale, globular, wrinkled surface and showed discrete cortical petechiae

![Gross anatomy findings in the kidneys of the cat with ethylene glycol intoxication](image)

**Fig. (7):** Gross anatomy findings in the kidneys of the cat with ethylene glycol intoxication

![Atrophied left kidney due to ethylene glycol toxicity.](image)

**Fig. (8):** Atrophied left kidney due to ethylene glycol toxicity.

![Longitudinal through kidney 5 days after ingestion of ethylene glycol showing dilated renal pelvis and bulged parenchyma from cut surface.](image)

**Fig. (9):** Longitudinal through kidney 5 days after ingestion of ethylene glycol showing dilated renal pelvis and bulged parenchyma from cut surface.
5. DISCUSSION:

Ethylene glycol (EG) is considered to be a highly nephrotoxic agent which found in antifreeze and other industrial solvents. Cats are more susceptible to toxic effect of Ethylene glycol toxicity than dogs. It is quickly absorbed from the digestive tract and the blood concentration reaches the peak within 1-4 hours after ingestion and almost all of the toxin is excreted or metabolized within 18-24 hours after ingestion (Oehme et al. 2000).

There were many clinical signs were observed in ethylene glycol toxicity in cats under experiment as lack of appetite, anorexia, vomiting, Oral ulcers/sores on mouth, Salivation, Lethargy, low body temperature, producing only small amounts of urine, large painful kidneys on palpation, oliguria and death may occurred which agree with clinical signs described by Crowell et al. 1979. Decreased body temperature in cats under the experiments is attributed to depressant effect of EG while the anorexia, convulsions and coma could be caused as a result of uremia which agrees with Paton, 1989. Increased pulse rate may be attributed to acidosis which agrees with findings of Herd, 1992. Recumbency and death of affected cats may be caused by hypocalcemia, azotemia and metabolic acidosis which agreed with Crowell et al. 1979.

Regarding to the biochemical serum analysis of examined cats under the experiment, our results showed that, the BUN and creatinine were significantly increased at the end of the experiment which occur as a result of renal damage. This agreed with Herd., 1992. Sigh et al. 1995 who reported that the hypovolemia that caused by dehydration leads to impaired excretion of urea and creatinine results in increasing their levels in the circulation.

Serum calcium level was significantly increased after EG ingestion which could be attributed to the formation of calcium oxalate as a result of binding of calcium to glyconate end product of ethylene glycol metabolism to form calcium oxalate crystals, while serum phosphorus level was significantly decreased as a result of the decreased renal excretion of phosphorus which described by Sigh et al. 1995. It is important to note that clinical signs of hypocalcemia do not occur due to the shift of the active, ionized calcium as a result of acidosis which mentioned by Thrall et al. 1984.

The serum sodium levels was significantly increased after EG ingestion as compared with its level before starting the experiment, this could be attributed to the dehydration which agreed with Sigh et al. 1995. On contrary, the serum chloride showed a significant decrease after EG ingestion, which agreed with Kaneko, 1989 who attributed that to loss of the chloride in urine as a result of tubular defect in case of renal disorders so its level in the circulation become low.

The serum level of potassium was significantly decreased after EG ingestion and this agreed with Carlson, 1989 who reported the hypokalemia as a result of excess mineralocorticoid and altered renal tubular function due to tubular acidosis or postobstructive stages.

Hematological analysis of blood of cats under experiment showed that there was a significant increase in RBCs count while Hb content was significantly decreased especially at 5th day, while PCV significantly increased at the end of the study, this may attributed to
reduced production of erythropoietin, a renal hormone that controls the bone marrow’s production of red blood cells, as kidney disease progresses. (Chahoub et al, 2011).

Regarding to the WBCs level, blood plateles, neutrophils and lymphocytes, there was a significant increase in their levels after EG administration as compared with their levels before starting of the experiment which agree with Thrall et al. 1984. Leucocytosis in case of ethylene glycol intoxication may be caused by uremia as mentioned by Coles, 1974.

Urinalysis of cats after EG ingestion, showed isothenuria which could be attributed to osmotic diuresis and hyperosmolarity-induced polydipsia, hematuria and glucosuria as mentioned by Connally et al., 2010.

Calcium oxalate crystals could appear in the urine within 3-6 hours after ingestion in which oxalate bind to plasma calcium forming calcium oxalate crystals in the renal tubules which greed with Oehme et al. 2000.

Regarding to ultrasonongraphic examination of kidneys of cats under the experiment, it showed enlarged kidneys, with increased cortical echogenicity due to accumulation of calcium oxalate crystals, and enhanced corticomedullary distinction and this agree with (Ettinger, and Feldman, 2010).

The P.M examination of kidneys revealed that they were enlarged and of a cloudy greyish color. The capsule was easily removed, and the boundary between cortex and medulla was clearly demarcated. The cortex was spattered with grey-yellowish bands and among them, with grey-whitish granular structures, barely visible to the naked eye and this agree with (Rowland, 1987).

In conclusion, ethylene glycol (antifreeze) is considered as one of the most life-threatening toxins encountered in small veterinary medicine and precautions should be applied to prevent animals to reach it. It could cause serious injuries to kidney, liver, CNS and acid base balance. Therefore, emergency treatment should be initiated rapidly within 8 hours of ingestion to prevent the progression of acute kidney injury.

REFERENCE:


