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Monensin Toxicosis in Camels Reared in Egypt: Updating Clinical and Clinicopathological Investigations

*Mousa S. A. and El-Hamamsy H. T.*

1Department of Internal Medicine and Infectious Disease, Faculty of Veterinary Medicine, Cairo University, Giza, Egypt.

**Abstract**

An acute and sub acute onset of illness were reported in a camel ranch in Egypt after accidental feeding a claimed newly imported broiler finisher concentrate containing ionophore monensin sodium with concentration (100mg/kg feed), So this work was carried out to study the alterations in clinical and clinicopathological pictures in victim camels with acute illness (6) and sub acute illness (9). Clinical observations were recorded and blood and urine samples were taken from all victim camels for clinicopathological analysis. Victim camels showed signs of acute heart failure with significant increase in respiratory rate, heart rate, muscular tremors, staggering and falling with lateral recumbancy in acute cases while sub acute cases showed signs of congestive heart failure, S.C edema in the area extended from the prepuce toward umbilicus and varying degree of myoglobulinuria. Clinicopathological picture showed significant elevations in PCV, AST, CPK and LDH (p≤0.001), hypocalcaemia, and hyperphosphatemia i.e disturbance in Ca: P ratio..Urinalysis revealed severe myoglobinuria with chocolate brown discoloration of urine in acute toxicosis and red brown discoloration in sub acute cases. We can concluded that acute and sub acute monensin toxicosis in camels characterized by signs of heart failure, elevation in serum muscle enzymes, hemoconcentration and myoglobinuria.

**Keywords:** Monensin, toxicity, camels, clinical, clinic-pathological, Egypt.
Introduction

Monensin is a polyether ionophore antibiotic produced by Streptomyces Cinnamomones used as feed additive to improve growth and feed efficiency and control ketosis in cattle as well as coocidicidal in poultry industry (Divers et al., 2009). It produces lipophilic complex with cations (Ca++, K+, Mg++) which facilitate their transport via biological membranes (EMEA 2007 and Divers et al., 2009). Monensin has a small therapeutic margin in target and non-target species, toxic syndromes occur as a result of inappropriate use or accidental contaminations of ration (Bila et al., 2001). Monensin toxicity primarily affect cardiac and skeletal muscles as recorded by (Grooms, 2010) in horses and (Hernandez et al., 2012) in feedlot cattle. Signs of toxicity were similar in all animals as anorexia, hypo activity, skeletal muscle weakness, ataxia and diarrhea as recorded by (Dalvi et al., 1990) in goats, (Decloedt, 2012 and Vagra et al., 2013) in horses and (Hernandez et al., 2012 and Vagra and Puschner, 2013) in cattle. The reported oral LD50 values (mg/kg b.w) in laboratory species were: rats 21.7 to 50, dogs >10 (female) to >20 (male) while in non-laboratory species were: horses 1.3 to 3, pigs 17 to 50, cattle 22 to 80, chicken 130 to 250 while in camels not recorded (EMEA, 2007). Horse is the most sensitive species to monensin toxicity while buffaloes have a lower tolerance to monensin toxicity than cattle (EFSA, 2008). Toxic cellular effects of monensin specially at the level of skeletal and cardiac muscle depend on dose and route of administration (Huczynski and Lowicki, 2013). There are scarce information on monensin toxicosis in camels across literature, So the present study was carried out to investigate the clinical and clinicopathological changes in victim camels exposed to accidental monensin toxicity.

Material and Methods

Animals

A total number of twenty two victim dromedary camels belonging to private camel ranch (with total 100 camel) located at the desert road near Cairo. These camels were stalled freely and fed on 4kg of concentrate /head daily, green fodder, fresh water and common salts were provided ad lib. The problem of sudden death and acute illness were noticed 2-3 days after feeding a newly imported broiler finisher concentrate containing monensin with concentration (100mg/kg feed) this ration was manufactured by PURINA ITALIA SpA. The camels were divided according to presence or absent of signs and severity, onset and duration of illness into 3 groups: The first group G1 (n=6) acute illness group with signs of circulatory and respiratory distress, muscular tremors, foamy nasal discharge, staggering and falling with lateral recumbancy with no response to external stimuli. These camels were emergency slaughtered after 1-2 days. The second group G2 (n=9) sub acute illness group with signs of restlessness, depression, pseudo - paralysis involving hind quarter, varying degree of myoglobinuria and dark colored foul smelling diarrhea were noticed in both groups. Third group G3 (n=7) control group which are apparently healthy camels and not exposed to the accidental ingestion of the monensin. All camels were exposed to complete and comprehensive clinical examination including (respiratory rate, pulse rate, heart rate and rectal temperature) as the method described by (Radostits, 2010). The dead (n=3) or slaughtered camels (n=3) exposed to postmortem examination.

Samples

From each camel two peripheral blood samples were collected from the jugular vein one into EDTA treated tubes were used to establish cellular blood constituents according to (Sharma and Singh, 2000). The other into a clean dry centrifuge tube for collection of clean non – hemolyized serum for determination of serum concentration of AST, CPK, LDH, and inorganic phosphorus levels (Spinreact company, Spain), creatinine (Bio-Diagnostic, Giza, Egypt), sodium, potassium (TECO - diagnostics company, U.S.A.), calcium (Spectrum company, Egypt) and BUN and glucose levels ( Biosystems company, Spain) on a specific spectrophotometer (Apple 302, USA). Urine samples were collected via urination into clean plastic containers for urinalysis using Medi-Test Combi 10 SGL (MACHEREY –NAGEL, France).
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Statistical Analysis

The obtained data were expressed as mean and standard error of the mean (mean ±SE) and analyzed statistically by SPSS program Version 16 using T- test. Significant differences in the values between the acute, sub acute group and control group were indicated by P*≤0.05, P*≤0.01 and P***≤0.001.

Results

Camels with acute illness showed significant increase in respiratory rate and its depth , marked elevation of heart rate with tachyarrhythmia, pulse rate was rapid and weak and temperature was in normal range. While camels with sub acute illness showed changes lower than acute group. Morbidity rate was 1.5% , mortality rate was 3% and case fatality 20%. These results confirm that camels are sensitive to monensin toxicity.

Hematological analysis (Table 1) revealed significant increase in PCV (P≤0.001)and in Rbcs (P≤0.01) in both groups that indicating severe hemoconcentration and significant increase in leucocytes(P≤0.001) in acute group Serum biochemical analysis( Table 2) revealed that acute cases showed significant increase in BUN, K and P levels (P≤0. 01) and significant reduction in Na and Ca levels (P≤0.001) , While sub acute cases showed significant increase in creatinine and muscle enzymes(P≤0.001) with significant reduction in glucose and Na levels(P≤0.001) . Urinalysis (Table 3) showed variable changes which were generally correlated with the severity of toxicosis. Myoglobinuria was a consistent findings and provide further evidence of myopathy.

Table 1: Hematological parameters in control and victim camels (with SI units).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>G1 (n=6)</th>
<th>G2 (n=9)</th>
<th>G3 (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBCs (million/cumm)</td>
<td>13.90 ± 0.2 **</td>
<td>13.70 ± 0.2 **</td>
<td>12.32 ± 0.3</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>43.70 ± 0.2</td>
<td>38.50 ± 0.4</td>
<td>32.90 ± 1.1</td>
</tr>
<tr>
<td>Hb (%)</td>
<td>12.90 ± 0.3 **</td>
<td>12.75 ± 0.5 **</td>
<td>11.65 ± 0.8</td>
</tr>
<tr>
<td>Total leucocytes (thousand/cumm)</td>
<td>11.10 ± 0.8 **</td>
<td>9.50 ± 0.8 **</td>
<td>6.68 ± 0.3</td>
</tr>
</tbody>
</table>


Table 2: Serum biochemical parameters in control and victim camels (with SI units).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>G1 (n=6)</th>
<th>G2 (n=9)</th>
<th>G3 (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN (mmol/l)</td>
<td>20.22 ± 2.4 ***</td>
<td>13.73 ± 2.2 ***</td>
<td>8.17 ± 1.6</td>
</tr>
<tr>
<td>Creatinine ( mmol/l)</td>
<td>347.41 ± 20***</td>
<td>295.25 ± 2.0 **</td>
<td>239.65 ± 12</td>
</tr>
<tr>
<td>Glucose ( mmol/l)</td>
<td>3.49 ± 0.2 ***</td>
<td>2.80 ± 0.1 ***</td>
<td>4.39 ± 0.3</td>
</tr>
<tr>
<td>AST ( U/L)</td>
<td>&gt; 2015</td>
<td>900 ± 87.3 ***</td>
<td>95 ± 3.2</td>
</tr>
<tr>
<td>CK (U/L)</td>
<td>&gt; 5000</td>
<td>2600 ± 12.2 ***</td>
<td>75 ± 2.1</td>
</tr>
<tr>
<td>LDH (U/L)</td>
<td>&gt; 3000</td>
<td>1200 ± 98 ***</td>
<td>290 ± 8.2</td>
</tr>
<tr>
<td>Ca (mmol/l)</td>
<td>0.75 ± 0.2 ***</td>
<td>1.41 ± 0.4 ***</td>
<td>2.41 ± 0.3</td>
</tr>
<tr>
<td>P (mmol/l)</td>
<td>3.29 ± 0.3 ***</td>
<td>2.79 ± 0.5 ***</td>
<td>1.79 ± 0.2</td>
</tr>
<tr>
<td>Na ( mEq/L/L)</td>
<td>95 ± 3.1 ***</td>
<td>106 ± 4.2 ***</td>
<td>135 ± 2.7</td>
</tr>
<tr>
<td>K (mEq/L/L)</td>
<td>5.6 ± 0.4 ***</td>
<td>4.8 ± 0.8 ***</td>
<td>3.8 ± 0.2</td>
</tr>
</tbody>
</table>


Table 3: Urinalysis in control and victim camels.

<table>
<thead>
<tr>
<th>Test</th>
<th>G1 (n=6)</th>
<th>G2 (n=9)</th>
<th>G3 (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>Chocolate brown</td>
<td>Red brown</td>
<td>Light–dark yellow</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>PH</th>
<th>Specific gravity</th>
<th>Occult blood</th>
<th>Ketons</th>
<th>Bilirubin</th>
<th>Urobilinogen</th>
<th>Glucose</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6.1 ± 0.5</td>
<td>1.052</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td></td>
<td>6.5 ± 0.2</td>
<td>1.050</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td></td>
<td>7.7 ± 0.4</td>
<td>1.029</td>
<td>N.S</td>
<td>N.S</td>
<td>N.S</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>

G1= Acute group  G2= Sub acute group  G3= Control group  N.S= non significant change.

Discussion

Outbreaks of toxicosis should be suspected when number of healthy animals were affected at the same time and showing the same signs and necropsy findings with previous history of feeding of a new component or change of ration or after medication. In our study on the basis of case history and epidemiological investigations there were not any contagious disease except, the breeders fed his camels in claimed ration of broiler containing monensin with concentration (100mg/kg feed). Although the literatures contained several studies about monensin toxicity in other animals like cattle, sheep and horses but it seems rare in camels. So our clinical observation and laboratory examination were discussed as following.

It was known that accidental ingestion of feed intended for chickens containing monensin at maximum authorized level of 120 and 125 mg /kg feed respectively, presents a health risk for several non – target species as camels (EFSA, 2008). In our victim camels exposed to 100mg/kg feed i.e 1-2 mg /kg b.w in average caused acute and sub acute illness and toxicosis

Clinical signs and necropsy findings in acute and sub acute victim camels: Camels with acute illness showed severe cardiac and respiratory manifestations similar findings were recorded in sheep by Miller et al., (1990), in dairy cattle by Gonzalez et al., (2005) and Vagra and Puschner (2013) and in horses by Bila et al., (2001). These camels either dead suddenly or emergency slaughtered and necropsy findings were pulmonary congestion and edema with froth formation in the air passage, heart had scattered dark areas of epicardial hemorrhage and areas of ventricular myocardial polar similar findings were observed in horse by Bila et al., (2001) and in cattle by Hernandez et al., (2012) and Vagra and Puschner (2013). These results confirm that the main cause of death in camels found dead was congestive heart failure induced by myocardiopathic effect of monensin toxicosis similar results were recorded in horse by Bila et al., (2001) and in cattle by Hernandez et al., (2012) and Vagra and Puschner, (2013).

Camels with sub acute illness showed dilatation and engorgement of superficial blood vessels and S.C edema in the area extended from the prepuce toward umbilicus similar findings were recorded in cattle by Hernandez et al., (2012) Fig (1). Varying degree of skeletal myopathies and myoglobinuria particularly in hind limbs that swollen, hard painful and oozing red brown discharge on puncture Fig (2) the same findings were recorded in sheep by Miller et al., (1990) in dromedary camels by Mousa et al., (1992) in dairy cattle by Gonzalez et al., (2005) and in horses by Rothwell (2010). The necropsy findings were flabby heart with visible myocardial polar and bilaterally symmetrical muscle damage with whitish gray calcification the same results were noticed in cattle by Hernandez et al., (2012) and Vagra and Puschner, (2013). These results confirm that the main cause of illness in camels was congestive heart failure induced by myocardiopathic effect of monensin toxicosis Clinicopathological and urinalysis in acute and sub acute victim camels: Hematological analysis revealed significant increase in PCV (P≤0.001) and in Rbcs (P≤0.01) in both groups indicating severe hemococoncentration the similar result were recorded in sheep by Miller et al., (1990) in dromedary camels by Mousa et al., (1992). In dairy cattle by Gonzalez et al., (2005) and in horses by Rothwell (2010) this may be due to acute tubular
nephrosis as reported by Langston et al., (1985). The significant increase in leucocytes (P≤0.001) may reflect a response to monensin toxicosis. Serum biochemical analysis revealed that acute cases showed significant increase in BUN, K and P levels (P≤0.01) these results similar to findings reported in bacterian camels by Miller et al., (1990) and Gonzalez et al., (2005) in dairy cattle. This increases in sub acute cases may be due to circulatory insufficiency with decreased glomerular filtration while in acute cases the marked increase may be due to circulatory failure and tubular damage. Significant hyperglycemia occur as a result of stress Activities of the enzymes AST, CPK and LDH revealed highly significant increase (P≤0.001) as the result of severe skeletal and cardiac muscle damage in severely affected camels. Similar result were recorded in sheep by Miller et al., (1990), in horse by Bila et al., (2001) and in buffaloes by Rozza, et al., (2007). Our result revealed that significant hyponatremia (P≤0.001) similar result was recorded in sheep by Miller et al., (1990) and in dairy cattle by Gonzalez et al., (2005). This may be due to monensin enhance selective ion transport for (Na, K). Also significant hypocalcaemia (P≤0.001), significant hyperphosphatemia (P≤0.001) and significant hyperkalemia (P≤0.001) were recorded in our results.

Uranalysis showed variable changes which were generally correlated with the severity of toxicosis. Myoglobinuria was a consistent finding and provide further evidence of myopathy. The degree myoglobinuria depended upon the pattern of muscular damage.

Light myoglobinuria i.e red –brown discoloration was associated with sub acute toxicosis, and intense myoglobinuria i.e chocolate – brown discoloration was associated with acute toxicosis Fig (3), similar findings were recorded in calves, dogs, horses and swine few days after exposure by (Van Vleet, 1987, Langston et al., 1983), Bila et al., (2001) and Novilla, (2007) respectively. Urine specific gravity was higher in acute cases it may be due to decreased urine out put caused by circulatory failure and decreased glomerular filtrate.

Decreased urine PH generally correlated with occurrence of myoglobinuria. Detection of glucose and protein in the urine of affected camels may be due to the tubular damage and decreased reabsorption in the kidneys. No significant changes in urine ketone, bilirubin and uroblinogen were detected in victim camels.

### Clinical Signs and Necropsy Findings in Acute and Sub Acute Victim Camels

Camels with acute illness showed significant increase in respiratory rate and its depth, marked elevation of heart rate with tachyarrhythmia, pulse rate was rapid and weak and temperature was in normal range similar finding were recorded by (Vagra and Puschner, 2013) in cattle. While camels with sub acute illness showed changes lower than acute group.

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Clinico-Pathological and Urinalysis in Acute and Sub Acute Victim Camels

Hematological analysis revealed significant increase (P**≥0.01) in Rbcs and Hb % indicating sever hemoconcentration the similar result were recorded in sheep by (Miller et al., 1990) in dromedary camels by (Mousa et al., 1992) in dairy cattle by (Gonzalez et al., 2005) and in horses by (Rothwell, 2010) this may be due to...
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Urinalysis showed variable changes which were generally correlated with the severity of toxicosis. Myoglobinuria was a consistant findings and provide further evidence of myopathy. The degree myoglobinuria depended upon the pattern of muscular damage. Light myoglobinuria i.e red – brown discoloration was associated with sub acute toxicosis, and intense myoglobinuria i.e chocolate – brown discoloration was associated with acute toxicosis Fig (3), similar findings were recorded in calves, dogs, horses and swine few days after exposure by (Van Vleet, 1987, Langston et al., 1983, Bila et al., 2001 and Novilla, 2007) respectively. Urine specific gravity was higher in acute cases it may be due to decreased urine out put caused by circulatory failure and decreased glomerular filtrate. Decreased urine PH generally correlated with occurance of myoglobinuria. Detection of glucose and protein in the urine of affected camels may be due to the tubular damage and decreased reabsorption in the kidneys. No significant changes in urine keton, bilirubin and urobilinogen were detected in victim camels.

**Conclusions**

Out study consider the first report of monensin toxicity in Egyptian camels and confirm that camels are sensitive to monensin toxicity. Acute and sub

![Image](image_url)
brown discoloration in acute cases and red – brown discoloration in sub acute cases

Acknowledgment

The authors are thankful to the animal owners for allowing to draw blood samples from their animals for use in the study. And very thankful to department of internal medicine and infectious diseases, Faculty of veterinary medicine, Cairo university, Egypt for supporting and funding this work.

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