Monensin Toxicosis in Camels reared in Egypt: updating clinical and clinicopathological investigations.

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Abstract: An acute and sub acute onset of illness were reported in a camel ranch in Egypt after accidental feeding a clamed newly imported broiler finsher concentrate containing ionophore monensin sodium with concenteration (100mg/kg feed), So this work was carried out to study the alterations in clinical and clinic-pathological pictures in victim camels. Victim camels showed signs of acute heart failure with significant increase in respiratory rate and its depth, marked elevation of heart rate with tachyarrhythmia, pulse rate was rapid and weak, muscular tremors,respiratory distress, staggering and falling with lateral recumbancy in acute cases while subacute cases showed signs of congested heart failure, restlessness, depression, S.C edema in the area extended from the prepuce toward umbilicus and varying degree of myoglobulinuria and dark colored foul smelling diarrhea were noticed in both groups. Clinico-pathological picture showed significant elevations ($p \leq 0.001$) in PCV, muscles enzymes (AST, CPK, LDH), hypocalcaemia, hyponatremia, hypokalemia and hyperphosphatemia. Urinalysis revealed sever myoglobinuria with chocolate brown discoloration of urine in acute toxicosis and red brown discoloration in sub acute cases.

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Introduction

Monensin is a polyether ionophore antibiotic produced by Streptomyces Cinnamonesis used as feed additive to improve growth and feed efficiency and control ketosis in cattle as well as coocidiocidal in poultry industry. It produce lipophilic complex with cations (Ca^{++}, K^{+}, Mg^{++}) which facilitate their transport via biological membranes (EMEA,2007). 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, signs of toxicity were similar in all animals as anorexia, hypoactivity, skeletal muscle weakness, ataxia and diarrhea. The reported oral LD_{50} 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). There were little studies on monensin toxicosis in camels across literature, So the present study was carried out to investigate the clinical and clinico-pathological changes in camels exposed to accidental monensin toxicity.

Materials and Methods

Animals:

A total number of twenty two victim dromedary camels belonging to private camel ranch located at the desert road near Cairo. These camels were stalled freely and fed on 4kg of concentrate /head daily, green fooder, 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 finsher concentrate containing monensin with concenteration (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, straggering 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 third quarter, varying degree of myoglobulinuria and dark colored foul smelling diarrhea were noticed in both groups. Third group G3 (n=7) control group which are apparently healthy camels. 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 (Radostitis,2010). The dead or slaughtered camels 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 Schalm (1986). The other into a clean dry centrifuge tube for collection of clean non –heamoliyzed 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 maturation into clean plastic containers for urinalysis using Medi-Test Combi 10 SGL(MACHEREY –NAGEL, France).

Statistical analysis:

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

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.

Table (1): Hematological parameters in control and victim camels

Parameters	**G1 (n=6)**	**G2 (n=9)**	**G3(n=7)**
Rbcs ($\times 10^6$/ml)	13.90 ± 0.2**	13.70 ± 0.2**	12.32 ± 0.3
PCV (%)	43.70 ± 0.2	38.50 ±0.4	32.90 ± 1.1
Hb(%)	12.90 ± 0.3**	12.75 ± 0.5**	11.65 ± 0.8
Total leucocytes($\times 10^3$/ml)	11.10 ± 0.8**	9.50 ± 0.8**	6.68 ± 0.3

G1= Acute group; G2= Sub acute group; G3= Control group.

Hematological analysis revealed significant increase($P^{**}\geq 0.01$) in Rbcs and Hb % indicating sever hemoconcenteration and significant increase ($P^{**}\geq 0.01$) in leucocytes**.**

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

Parameters	**G1 (n=6)**	**G2 (n=9)**	**G3(n=7)**
BUN (mg/dL)	57.2 ± 2.4***	38.47 ± 2.2***	22.9 ± 1.6
Creatinine(mg/L)	3.93 ±.20***	3.34 ±.20*	2.71 ±.12
Glucose(mg/dL)	68.46 ± 0.2***	50.45 ± 0.1***	79.27 ± 0.3
AST (U/L)	2015	900 ± 87.3***	95 ± 3.2
CK (U/L)	5000	2600 ± 0.12***	75 ± 2.1
LDH (U/L)	3000	1200 ±.98***	290 ± 8.2
Ca (mg/dL)	3.00 ± 0.2***	5.62 ± 0.4***	9.63 ± 0.3
P (mg/dL)	10.21 ± 0.3***	8.66 ± 0.5***	5.57 ± 0.2
Na (mEqL/L)	95 ± 3.1***	106 ± 4.2***	135 ± 2.7
K (mEqL/L)	5.6 ± 0.4***	4.8 ± 0.8***	3.8 ± 0.2

G1= Acute group; G2= Sub acute group; G3= Control group.

Serum biochemical analysis revealed that acute cases showed significant increase ($P^{***}\geq 0.001$) in BUN, Creatinine and glucose. Activities of the enzymes AST,CK and LDH revealed highly significant increase ($P^{***}\geq 0.001$) also significant hypocalcemia ($P^{***}\geq 0.001$), Significant hyperphosphatemia ($P^{***}\geq 0.001$) and significant hyperkalemia ($P^{***}\geq 0.001$) were recorded in our results.

Table (3) Urinalysis in control and victim camels

Test	G1 (n=6)	G2 (n=9)	G3 (n=7)
Color	Chocolate brown	Red brown	Light–dark yellow
PH	6.1 ± 0.5	6.5 ± 0.2	7.7 ± 0.4
Specific gravity	1.052	1.050	1.029
Occult blood	Myoglobin	Myoglobin	Nil
Ketons	N.S	N.S	Nil
Bilirubin	N.S	N.S	Nil
Urobilinogen	N.S	N.S	Nil
Glucose	Present (+++)	Present (++)	Nil
Protein	Present(+++)	Present(++)	Nil

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

Urinalysis showed variable changes which were generally correlated with the severity of toxicosis. Myoglobinuria was a consistant findings and provide further evidence of myopathy.

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 hi scamels in clamed ration of broiler containing monensin with concenteration (100mg/kg feed). Although the literature 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 groups:

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

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 Figure (1). Varying degree of skeletal myopathies and myoglobinuria particularly in hind limbs that swollen, hard painful and oozing red brown discharge on puncture Figure (2) the same findings were recorded in sheep (Miller et al, 1990), in dromedary camels (Mousa et al, 1992),in dairy cattle (Gonzalez et al, 2005) and in horses (Rothwell, 2010). The necropsy findings were flabby heart with visible myocardial pollar and bilaterally symmetrical muscle damage with whitish gray calcification. These result confirm that the main cause of illness in camels was congestive heart failure induced by myocardiopathic effect of monensin toxicosis.

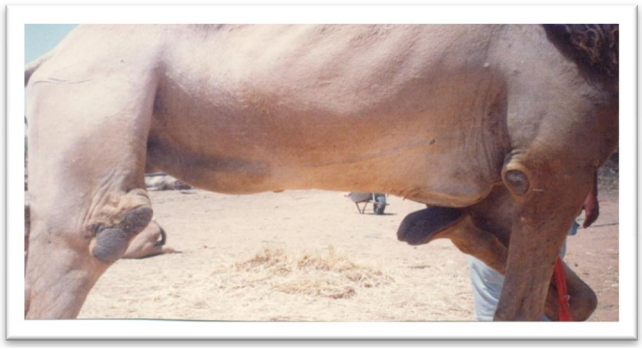
Clinico-pathological and urinalysis in acute and sub acute victim camels:

Hematological analysis revealed significant increase ($P^{**} \leq 0.01$) in Rbcs and Hb % indicating sever hemoconcenteration the similar result were recorded in sheep (Miller et al, 1990), in dromedary camels (Mousa et al, 1992), in dairy cattle (Gonzalez et al, 2005) and in horses (Rothwell, 2010) this may be due to acute tubular nephrosis as reported by (Langston et al, 1985). The significant increase ($P^{**} \leq 0.01$) in leucocytes may reflect a response to monensin toxicosis.

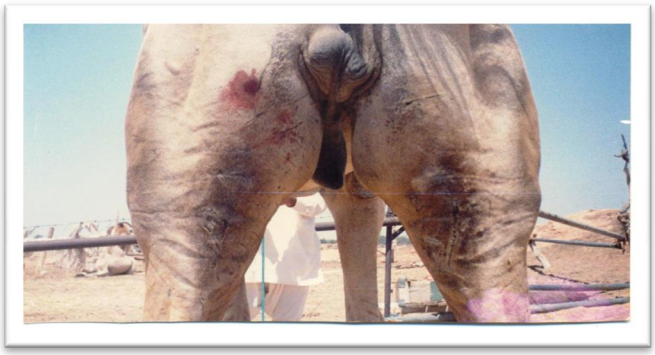
Serum biochemical analysis revealed that acute cases showed significant increase ($P^{***} \leq 0.001$)

in BUN, Creatinine and glucose levels these results similar to findings reported in bacterian camels by (Miller et al, 1990) and (Gonzalez et al, 2005) in dairy cattle except in glucose level. 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.

Figures



he area extended from the prepuce toward umbilicus



athies particularly in hind limbs that swollen, hard

Figure (3): Urine samples from normal camels(straw yellow discoloration), camels with sub acute toxicosis(red – brown discoloration) and camels with acute toxicosis(chocolate – brown discoloration).

Activities of the enzymes AST,CK and LDH revealed highly significant increase ($P^{***}\leq 0.001$) as the result of sever skeletal and cardiac muscle damage in severely affected camels. Similar result were recorded by (Miller et al, 1990) in sheep and (Bila et al, 2001) in horse. Our result revealed that significant hyponatremia ($P^{***}\leq 0.001$) similar result was recorded by (Miller et al, 1990) in sheep and (Gonzalez et al, 2005) in dairy cattle this may be due to monensin enhance selective ion transport for (Na, K). Also Significant hypocalcemia ($P^{***}\leq 0.001$), Significant hyperphosphatemia ($P^{***}\leq 0.001$) as the result recorded by(Miller et al, 1990) and significant hyperkalemia ($P^{***}\leq 0.001$) were recorded in our results.

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 similar findings were recorded in dogs, calves, horses and swine few days after exposure (Langston et al, 1985, Van Vleet, 1983, Bila, 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.

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