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Short Communication

High mortalities among one-humped camels (*Camelus dromedarius*) due to salinomycin poisoning in the Kingdom of Saudi Arabia

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ABSTRACT

Objective: The objective of this study was to investigate the cause of death of large number of camels during an outbreak in Saudi Arabia.
Material and methods: History was taken from the camel owners and breeders. Besides, clinical and post-mortem (PM) examinations were conducted. In this study, ten locations were surveyed and all camels were examined. Wheat bran was suspected as the source of the havoc. For establishing this assumption, a feeding trial was conducted with three camels, six mice, one rabbit and four of each chickens and ducklings using the incriminated wheat bran. Samples were collected from the suspicious wheat bran and the afflicted animals, and were sent to international reference laboratories for diagnosis. The clinical signs elicited by the feeding trial were compared with the signs recorded in the outbreak.
Results: The body temperature of the affected camels ranged from 36.4°C to 41.9°C. The clinical signs included hyper-excitability, muscle tremors, in-coordination of the hind quarters, sternal or lateral recumbence, inability to stand, and death. PM examination revealed no remarkable pathological changes in internal organs but the rumens were full of gases, and showed hyperemia and petechial hemorrhages. Within a period of twelve days from the onset of the crisis, 2,800 of the affected camels died. The clinical signs showed by the two camels in the feeding trial were similar to those observed in field outbreak. The tentative diagnosis of toxicosis, which was made based on the clinical signs was confirmed by the reference laboratories. Salinomycin (300 to 400 mg/Kg feed), Aluminium (230 ppm), *Aspergillus clavatus* and *A. flavus* were detected in the incriminated wheat bran.
Conclusion: Salinomycin causes heavy mortalities in one-humped camels in the affected areas. Owners and breeders are adviced to avoid feeding low quality feed to their camels.

KEYWORDS

Camels; High mortalities; Wheat bran; Salinomycin; Saudi Arabia

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INTRODUCTION

Salinomycin and other carboxylic ionophores like monensin, lasalocid, narasin, and maduramicin are often used as anti-coccidial drugs in poultry and as growth and production promoters in ruminants because of their ability to alter gut flora, and therefore, improve nutrient absorption, daily weight gain in beef and milk production in dairy cattle (Story and Doube, 2004; Omidi et al., 2010).

Like other ionophores, over-dosage of salinomycin in non-target animal species might result in toxicosis (Plumlee et al., 1995). Such toxicity has been reported in farm animals like sheep, cattle, calves, rabbits and in pets (dogs and cats) and poultry such as chicken and turkeys (Peixoto et al., 2009; Pakozdy et al., 2010; Hosseini et al., 2013). In addition, it was also reported in pigs and horses (Konstanz et al., 1995; Van der Lide-Sipman et al., 1999; Pakozdy et al., 2010; Omidi et al., 2010).

Salinomycin toxicity in camels, alpacas, and bactrian, with varying case-fatality rates, a range of different clinical signs and economic losses, has also been reported (Murray and Miller, 2008; Al-Wabel, 2012; Mousa and El-Hamamsy, 2013). Moreover, salinomycin toxicosis has been reported in human beings (Story and Doube, 2004).

Accidental salinomycin intoxication with insidious clinical signs and high case-fatality rates in camels occurred after the consumption of poultry feed instead of pure wheat bran which is routinely fed to camels. In this study, the high mortalities of one-humped camels were investigated, and the clinical features described by the owners of the camels were verified experimentally by close observation of the clinical signs menifested in the experimental camels fed on the suspected poultry feed in which salinomycin was determined.

MATERIALS AND METHODS

History of the outbreak: In a farm in the Kingdom of Saudi Arabia (KSA), where poultry and 100 camels were kept in close vicinity for egg, meat, and milk production. The farm manager claimed that 40 camels were fed with poultry feed. Shortly after that, the camels showed various clinical signs followed by death within 24 to 48 h. The clinical signs included restlessness (frequent sitting and standing), hyper-excitability, salivation, lachrymation, sweating, in-coordination of the hind legs, staggering, sternal recumbence, inability to stand on the hind legs "as if paralyzed", extension of the neck and resting the head on the ground, lateral recumbence, arching of the neck, regurgitation of rumen contents through the mouth followed by death. A similar case happended during August 2007 in Wadi-Eldwasir where a large number of camels died within 20 min to 5 h after onset of different clinical signs; the possible cause was feeding wheat bran. Camels within herds in close vicinity of the affected herds were quite healthy and grazed normally (**Figure 1A**).

Drinking water was given to the camels from safe sources, and the same water was given to other healthy camels. No toxic plant was seen in and around the area of affected camels. Camel owners and breeders also reported that some shepherds experienced discomfort, itching and irritation on their hands during preparing the animal feed. Ten locations in Riyadh, Mecca, Asir, Najran, and Jazan were surveyed and the affected camels were treated symptomatically to alleviate the clinical signs observed.

Clinical and laboratory investigations: In accordance to Jackson and Cockcroft (2002), detailed history was taken along with visual inspection and physical examinations. Symptoms, clinical signs and physical parameters were recorded.

Post-mortem examination: Post-mortem (PM) examination of some dead camels was done. Samples comprising of whole blood, liver, spleen, intestines, rumen and/or rumen contents, kidneys, lungs and heart were collected. The samples were sent to the Toxicology, Hormone, and Drug Residues Laboratories of the Ministry of Agriculture, other governmental research and private laboratories in the KSA.

Tentative diagnosis: As a tentative diagnosis based on history, clinical signs shown by the affected camels including high mortalities, the wheat bran was suspected to be the source of an unknown toxic agent.

Feeding Trials

Ethical consideration: The experiment was conducted by following standard techniques considering camel welfare and ethical issues. The feeding trial was conducted to confirm the cause of the high mortalities of camels. The camels were reared in clean pens and cages, and were offered clean drinking water *ad libitum*.

Camels: The wheat bran was suspected to have the cause of the high mortalities. To reveal out this, 3 male camels aging between 27 and 32 months were purchased from the camel market during August, 2007. The animals were apparently healthy showing no clinical abnormalities; the camels were numbers from 1 to 3. Each camel was put in a separate pen. The feeding trial was conducted at the

National Agriculture and Animal Resources Research Centre, Riyadh, KSA.

Four sacks of wheat bran were collected from the owners and breeders who experienced high mortalities among their camels. The contents of the sacks were thoroughly mixed. The camel (1 and 2) were each provided with 7.5 Kg of the wheat bran. These two camels were fed once only. Camel number 3 was left as control provided with 7.5 Kg wheat bran that was purchased from Riyadh market after being inspected to ensure its purity.

Laboratory animals and birds: The feeding trial was also conducted in six mice, one rabbit and four of each chickens and ducklings. These laboratory animals and birds were put in clean cages in a well ventilated room, water was given *ad libitum*, and were fed on the suspicious wheat bran.

RESULTS

Clinical signs in camels and PM examination: Visual clinical examination of the affected animals revealed variable clinical signs including 85-90% mortality. Some camels were showing marked bloat and some were on lateral recumbency with arched neck resting the head on the ground. Others were on sternal recumbency with abducted hind quarters at the thigh region and legs pushed forwards (**Figure 1D**), and were unable to stand and reluctant to repel swarming flies (*Musca domestica*) from their face and around natural orifices. Some camels were found standing, dull and with soiled faces and flies gathering around the eyes, nostrils and mouth. The urine of some camels was reddish in color and the feces of some animals looked normal, but in others the fecal pellets were small in size, dry, light in weight and yellow to yellowish brown in color. Some camels appeared to have blurred vision and some were comatose. Other clinical signs comprised hyper-excitability, muscle tremors, in-coordination of the hind quarters, sternal or lateral recumbency and inability to stand followed by death.

Variable body temperatures were recorded ranging from 36.4°C to 41.9°C. The mucous membranes were pink rosette to pale in color. Percussion of the chest revealed dull, resonant or tympanic sounds. The symptomatic treatment that was given to the affected camels in the field outbreak produced only little and transient improvement, but soon after, the standing animals went down on sternal recumbency and were unable to stand. The condition of the animals on sternal recumbency became worse, went on lateral recumbency and died finally. Within a period of 12 days from the onset of the health problem, 2,800 of the affected camels died (**Figure 1E-F**).

PM examinations of a few dead camels revealed no remarkable pathological changes in internal organs but their rumen were full of gases, and showed hyperemia and petechial hemorrhages involving all internal organs including the meninges and brain.

Experimental feeding trials

Camels: Camel number 2 showed initial clinical signs after 50 min from feeding on the wheat barn while camel number 1 showed clinical signs after 5 h. The symptoms were restlessness; episodes of intermittent muscle tremors which started from the neck back to shoulders, flank, and hind quarters and disappeared in few seconds, followed by another episode. This clinical sign continued for 30 min. There was profuse lacrimation and contraction of the neck muscles and hind quarters followed by relaxation. This latter episode disappeared after 1 to 2 min, followed by another one which also continued for 20-30 min.

The experimental camels showed stampeding of the hind legs (indication of agony and pain), profuse sweating involving the axillae and flank and later on sweating involved the ventral aspect of the chest and abdomen. Camel 1 and 2 also showed crossing of the hind legs, staggering and in-coordination. They went down on sternal recumbency, had blurred vision, and were unable to stand on their hind legs (as if paralyzed). The body temperatures of both camels showed a lot of fluctuation but were both within the maximum normal range. Both camels went down on lateral recumbency with arched neck and rested their head on the ground. They became comatose with dropped lower lips. The mucous membranes were cyanotic and the two camels had rapid shallow respiration. Fluid ruminal contents was regurgitated through the mouth shortly before death. Terminally, camel number 2 died 36 h after the feeding trial, while camel number 1 showed marked loss of weight, became dull and died 5 days after feeding on the suspected wheat bran. Camel number 3, the control, survived until the end of the feeding trial and was free of any clinically detectable abnormality.

PM examination of the two camels (number 1 and 2) revealed severe pathological changes (**Figure 1G-I**). The meninges and brains of the animals were quite inflamed and showed petechial hemorrhages, and the blood vessels were engorged. The hearts were quite flabby, and the lungs were edematous, congested, collapsed in some areas and emphysematous in others. The livers were enlarged

Figure 1. Mortality of one-humped camel due to salinomycin poisoning. (A) camels are grazing on grass field, (B-C) salinomycin poison, (D) Sternal recumbacnce of camel due to salinomycin poisoning, (E) Lateral recumbancy of camel followed by dealth due to salinomycin poisoning, (F) Death of hundreds of camels due to salinomycin poisoning, (G-I) Post-mortem findings of salinomycin poisoning.

(*i.e.*, hepatomegaly), and showed white streaks and large hemorrhagic areas. The digestive tract was inflamed and showed petechial hemorrhages. The rumen was full of gas and the intestines were empty except for viscid greenish content in camel number 1 and fluid yellowish brown content in camel number 2. The spleen was dark and showed petechial hemorrhages. The kidneys were congested especially at the cortico-medullary junction. The urinary bladder in camel number 1 was empty, thick walled and inflamed, but the urinary bladder of camel number 2 contained 25-30 mL of dark hemorrhagic thick fluid.

Throughout the feeding trial, clotted and unclotted blood samples were regularly collected from the camels, after death, and at PM examination. Frozen and fixed tissues (10% formal saline), brains (12% buffered formal saline) and samples from the suspected wheat bran were sent to the Laboratoire de Toxicologie, Ecole Nationale Veterinaire de Lyon, France (contacted through the FAO Programme Coordination Office in Riyadh – by the first author [Chief Technicol Advisor]), to the Central Science Diagnostic Analysis Consultation Laboratories (IDAC), KSA, and to the Histopathology Laboratory, Ministry of Agriculture (MoA) Diagnostic Laboratories for investigations.

Laboratory animals and birds: The chicks and ducklings died shortly (24-36 h) after feeding on the wheat bran, the rabbit showed nervous signs and died 4 days afterwards, and the mice showed marked nervous signs but no mortalities were recorded.

Laboratory findings

Results on Salinomycin, *Aspergillus clavatus*, *A. flavus* and aluminium poisoning:

a. *Laboratoire de Toxicologie, Ecole Nationale Veteri-naire de Lyon, France*: Presence of *A. clavatus* which is usually associated with poisoning in animals affecting the nervous system causing muscle tremors, profuse salivation, protrusion of the eyes, in-coordination and

paralysis of the hind legs, congestion and emphysema of the lungs, hepatomegaly, occasionally kidney affection and death in 12-72 h.

b. *Central Science Laboratory (CSL), Sand Hutton, York, UK*: Presence of 300-400 mg/kg salinomycin in the wheat bran and presence of 559.2 ppm aluminium in the brain tissue of affected camels. They reported that examination of the incriminated wheat bran revealed the presence of hay particles, grass seeds, intact and ground maize grains, barley, stones, light brown material which resembled ground plant material of unknown identity and other substances. There was also a fine white powder which resembled starch or sugar (**Figure 1B-C**) which was stuck to the surface of the seeds and debris.

c. *Diagnostic Analysis Consultation Laboratories (IDAC)-KSA*: No pathogenic bacteria was isolated from organs, blood, rumen contents and feces of the affected camels. There was sharp increase in the blood platelets and drop in the percentage of hemoglobin. Serum enzyme analysis showed marked dysfunction of the liver and kidneys.

d. *Histopathology Laboratory, Ministry of Agriculture (MoA) Diagnostic Laboratories-KSA*: Hyaline degeneration and necrosis of the skeletal muscles of the shoulder and thigh, and hyaline degeneration of the myocardial muscles. The liver showed fatty degeneration (**Figure 1G-I**).

Final diagnosis: Based on the laboratory findings, the tentative diagnosis 'toxicosis' was confirmed, and salinomycin was identified as the main cause of the toxicity that resulted the death of 2,800 camels in a short period of time.

DISCUSSION

Animal poisonings with ionophore antibiotics, especially salinomycin, are widely described in different species with varying degree of morbidity and mortality (Murray and Miller, 2008). In the present outbreak, the preliminary diagnosis based on history and clinical examinations was confirmed by detecting salinomycin at 300 to 400 mg/Kg feed of incriminated wheat bran. Al-Nazawi and Homeida (2009) reported that an oral dose of salinomycin at 0.4 to 0.8 µg/Kg body weight (bwt) was enough to develop neurological signs and sero-biochemical alterations in camels. Another report by Al-Wabel (2012) showed that the effect of 15 mg/Kg bwt of salinomycin doses every second day in camels, would result in a significantly high death rate few days following the treatment. The amount of salinomycin reported herein is by far more than these amounts observed by Al-Nazawi and Homeida (2009) and Al-Wabel (2012). The high mortality reported herein was supported by several previous reports who found that the camels, alpacas, and bactrian were highly sensitive and susceptible to the intoxication with ionophore antibiotics (Al-Wabel, 2012; Mousa and El-Hamamsy, 2013). Furthermore, ionophore toxicity, often with case fatality as a result of overdosing, has recently been reported in camelids in different countries, and in species other than camels like dogs, cats, quail, chickens, rabbits, ostriches, goats, pigs, sheep, cattle and calves, and horses (Sawant et al., 1990; Andreasen and Schleifer, 1995; Plumlee et al., 1995; Hoop, 1998; Linde-Sipman et al., 1999; Bila et al., 2001; Agaoglu et al., 2002; Story and Doube, 2004; Aleman et al., 2007). Relatively low doses of ionphores can also be toxic to turkeys (Bartov, 1994; Van Assen, 2006; Novilla, 2007). In addition to animals, ionophore toxicity due to salinomycin has also been reported in human beings in New Zealand (Story and Doube, 2004).

The clinical signs of ionophore toxicity due to salinomycin varied from one animal species to another and were dose-dependent and were related organ/system involved in each species such as the musculoskeletal, cardiac, pulmonary, and nervous systems and smooth muscles. Even death may occur without any detectable clinical signs or symptoms (Murray and Miller, 2008).

The clinical signs observed in this outbreak authenticated the findings of Murray and Miller (2008), Al-Wabel (2012), and Hosseini et al. (2013), reporting that salinomycin caused toxicity and death. In the current outbreak, variable body temperatures were recorded (36.4 to 41.9°C) with mucous membranes ranging from pink rosette to pale in color. On percussion, dull, resonant or tympanic sounds were heard at the thoracic wall. The urine of the affected camels was reddish. Furthermore, nervous signs like blurred vision, coma, hyper-excitability, muscle tremors, and in-coordination of the hind quarters were observed.

The catastrophic case-fatality rate recorded in this outbreak was higher than those reported by Anderson (2003) and Murray and Miller (2008). Anderson (2003) recorded a fatality of over 50% in alpacas due to ionophore poisoning in Columbus, Ohio, USA among those animals fed solely on the contaminated concentrates and feed stuff, while Murray and Miller (2008) reported that the morbidity of salinomycin could reach up to 100% but the case fatality was approximately 50%. Mortalities due to salinomycin intoxication in animal species other than camels were also reported (Van Assen, 2006; Omidi et al., 2010; Hosseini et al., 2013).

In 2008, salinomycin intoxication was observed in a farm with 120 Holstein-Friesian cattle located in Birjand, Iran. Twenty-four calves of both sexes fed on a ration

containing a high concentration of salinomycin. This resulted in an overall mortality of 67%; 14 calves died within 10 days and 2 died within the following 60 days (Omidi et al., 2010). Turkey hens accidentally fed on broiler premix containing salinomycin sodium resulted in 34.5% mortality within a period of 9 days, in Manitoba, Canada (Van Assen, 2006). In the current outbreak, the high mortality among camels agreed with the findings of Al-Wabel (2012) who observed a mortality of 100% after a few days of treatment with salinomycin in a small clinical trial of dromedary camels and concluded that camels were quite sensitive to salinomycin.

Salinomycin intoxication causes recumbency and rising difficulties in camels and alpacas (Murray and Miller, 2008; Al-Wabel (2012). The lateral or sternal recumbency observed in this study with arching neck, abducted legs at the thigh region (**Figrue 1E**), and reluctant to expel flies from nose were agreed with the report of Van Assen (2006) who observed recumbency and prostration in turkey hens due to salinomycin intoxication. In the current outbreak, some of the affected camels were unable to stand on their hind legs. Others were staggering and showed incoordination of the hind legs. These observations were support with the findings of Murray and Miller (2008), Al Nazawi and Homeida (2009) and Al-Wabel (2012) who reported weakness, abnormal gait, ataxia, stiffness and recumbency. Neurological signs in calves resulting from salinomycin intoxication were also seen by Omidi et al. (2010) who described weakness, incoordindation and ataxia in affected calves.

Murray and Miller (2008), Al-Nazawi and Homeida (2009) and Al-Wabel (2012) observed symptoms that indicated the involvement of the gastrointestinal system, including changes in the quality and quantity of feces of camles in salinomycin intoxication. No remarkable pathological changes in any of the internal organs were found during the PM examination for three of the dead camels, except for congestion of the lungs in one camel and of all the brains, as reported by Anderson (2003).

Within twelve days of the current outbreak, many camels (n=2,800) died and the condition of the living ones became worse, as reported by Anderson (2003) who found similar outcomes in alpacas. PM examination of affected camels revealed ecchymosis and patches of jaundice in the subcutis, hyperemia and petechial hemorrhages involving all internal organs and the brain. The heart of dead animals was quite flabby and the lungs were edematous, congested and collapsed in some areas and emphysematous in other areas. The liver was enlarged, congested, and showed white streaks and large hemorrhagic areas and the kidneys were congested and showed petechial hemorrhages. The urinary bladder was thick walled and inflamed but empty, and the spleen was congested showing petechial hemorrhages, as reported by Murray and Miller (2008) and Al-Wabel (2012) who reported the involvement of the gastrointestinal, cardiac, pulmonary, and urinary systems and smooth muscles or death without detectable clinical signs or symptoms.

The clinical signs showed by the two camels in the feeding trial were identical to those observed in field outbreak after feeding on the incriminated wheat bran. The PM pictures were similar to those described by Murray and Miller (2008), Al-Nazawi and Homeida (2009), Omidi et al. (2010) and Al-Wabel (2012) in camels, alpacas, and claves.

The chicks and ducklings died shortly (24-36 h) after feeding on the wheat bran epitomizing the observations of Van Assen (2006) and Koutoulis et al. (2013). Moreover, our findings also authenticated the conclusions of Peixoto et al. (2009), who observed in a natural outbreak and verified experimentally that salinomycin toxicosis in rabbits resulted in nervous signs and death after few days, and the mice showed marked nervous signs but no mortalities were recorded.

Many different fungi were isolated from the wheat bran but *A. flavus* and *A. clavatus* were the only toxic species identified. However, *Aspergillus sp.* prefers high humidity and temperature, secretes many toxins and are usually associated with poisoning in animals affecting the nervous system causing muscle tremors, profuse salivation, protrusion of the eyes, in-coordination and paralysis of the hind legs, congestion and emphysema of the lungs, hepatomegaly, occasionally kidney affection and death in 12-72 h (McKenzie et al., 2004). The presence of *Aspergillus* in the wheat bran is highly indicative that the wheat bran has been exposed to high humidity during a certain period of time.

No pathogenic organisms were isolated from the organs, blood, rumen contents and feces of the affected camels. Moreover, the results obtained in the current outbreak and experimental camels are comparable to the findings of Al-Nazawi and Homeida (2009) and Al-Wabel (2012) who recorded a sharp increase in the blood platelets, significant increase in the total RBC count, drop in the percentage of hemoglobin and dysfunction of the liver and kidneys which are highly indicative of severe affection

A large amount of aluminium was found in the wheat bran and in the brain tissue of affected camels. The source of salinomycin was accidental due to feeding of

camels on poultry feed as the result of confusion. The source of aluminium was from the poor quality of the chemicals used for fumigation of wheat.

CONCLUSION

Salinomycin is toxic to camels and may lead to heavy economic losses (morbidities and mortalities), therefore, farmers, animal owners and breeders are strongly advised- (i) to buy feeds from authentic sources (feed factories or certified agents), (ii) to avoid stored feed after expiry date, (iii) to ensure storing animal feed under suitable conditions, (iv) to make sure that the stored wheat bran is normal in physical appearance and texture before it is fed to animals, (v) to avoid the storage of animal feeds near pesticides and chemicals, (vi) to use pesticides as directed by the manufacturers, and (vi) to consult with appropriate authorities in the Agriculture Branches in the different Governorates for extension services.

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The news of the tragic outbreak was widely circulated in the Local (KSA), Regional and International media for almost one year.

CONFLICT OF INTEREST

The authors declare no conflict of interests.

AUTHORS' CONTRIBUTION

Both the authors contributed equally.

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