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Pathological Studies on Camel Coccidiosis in the United Arab Emirates

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ABSTRACT

During the years 1996 and 1997, a total of 106 fresh camel carcasses from Dubai area were submitted for post-mortem examination including histological investigation. These animals included 20 calves (up to 100-kg body weight) and 86 older camels (28 young camels, 40 racing camels and 18 breeding camels). Forty-nine from a total of the 86 older camels were off food, emaciated and showed abdominal pain. The disease lasted between 1 to 14 days. Gross pathological examination of these cases revealed severe intestinal hemorrhages. Massive numbers of coccidian parasites of different stages and numerous eosinophilic granulocytes were detected in the lamina propria of the jejunum and ileum by histopathological studies. It was considered that these stages belong to *Eimeria (E.) cameli*, because typical immature oocysts of *E. cameli* were found within the intestinal epithelial mucosa. The 20 young calves were free of coccidias. The observed hemorrhagic-eosinophilic enteritis associated with a massive invasion of *E. cameli* suggests that this species is more pathogenic than in the literature suspected. To prove the pathogenicity of *E. cameli*, intestinal contents of 25 dissected camels suffering from coccidiosis were collected and the oocytsts were concentrated and sporulated. The oocysts needed 6 to 8 weeks to sporulate. Two young camels artificially infected with sporulated oocysts of *E. cameli* by the oral route excreted the parasite after 6 weeks (prepatency period).

Key Words:, Coccidiosis, Post-mortem, Camel, UAE.

INTRODUCTION

Regarding the importance of coccidiosis in camels, different opinions in the literature exist. Severe coccidiosis causing enteritis

and a mortality rate up to 10% in young camels have only been reported in a few cases (Hamanchadran *et al.,* 1968; Gruvell and Graber*,* 1969; Chineme, 1980; Levine, 1985; Hussein *et al.*, 1987; Kinne and Warnery*,* 1997).

On the other hand, many reports concerning the presence of coccidia oocysts in camels are based on investigations of fecal samples of healthy camels (Enigk, 1934; Boid *et al.,* 1986). Gruvell and Graber (1969) believe that coccidia in camels do not play an important role. This was due to the fact that out of 204 fecal samples examined only 14 were found to be positive for Eimeria. Gill (1976) found oocysts of *E. cameli, E. dromedarii, E. pellerdyi* and *E. bactriani* in 24% of fecal samples from dromedary camels in India. Mirza and Al-Rawas (1976) diagnosed 86% of fecal samples to be positive for E. dromedarii and E. noelleri oocysts in Iraq. Dubey and Pande (1964) identified *E. rajasthani, E. dromedarii* and *E. noelleri* oocysts in fecal samples from 45 healthy Indian camel calves. Yagoub (1989) detected 17.4% of a total of 230 fecal samples submitted from sudanese camels to be positive for coccidia oocysts.

A synergism might also exist between coccidia and the changed natural intestinal flora. Hamanchandran *et al.,* (1968) described fatal enteritis in camels caused by synergism between coccidias and Haemonchus longistipes. In chickens Baba *et al.,* (1997) reported that concurrent infection with *E. necatrix* and *C. perfringens* increases clostridial population in the intestine and has synergic effects on mortality and edema in the upper intestine.

The aim of this study was to check the incidence and importance of coccidiosis in our necropsy material, and to proof the pathogenicity of E. cameli in dromedary camels.

MATERIALS AND METHODS

During the years 1996 and 1997, a total of 106 fresh camel carcasses from Dubai area were submitted for post-mortem examination including histological investigation. These animals included 20 camel calves (up to 12 months old; group 1), 28 young camels (12 to 24 months old, group 2), 40 racing camels (2 to 12 years old, group 3) and 18 breeding camels (above 12 years, group 4). All necropsies were performed within 1 to 5 hours after death. Pieces of intestine, liver, spleen and lymph nodes were taken for microbiological investigations using routine methods. The intestinal

samples were also tested for the growth of anaerobes using Zeissler agar containing antibiotic supplement (Oxiod, SR93). The plates were incubated under anaerobic conditions (Gas generating kit, Oxoid) at 37°C for 48 hours. All intestinal samples were also spread onto Bacillus cereus agar containing egg yolk emulsion and Polymixin B supplement (Oxoid, SR99). The specimens were enriched in tetrathionate broth followed by culture on brilliant green phenol red lactose agar and pril mannitol agar for the detection of salmonella organisms.

For histopathological investigation small pieces of all parenchymatous organs, the gastrointestinal tract (compartments, abomasum, duodenum, jejunum, ileum, colon and caecum) as well as muscles and body lymph nodes were taken. From 25 camels suspected of coccidiosis samples from 15 locations of the small intestine (50-cm apart) were taken for histopathological investigations. These samples were fixed in 10% buffered formalin solution for 24 hours and 5 µm thin sections were cut and stained with hematoxilin and eosin (H&E-stain).

Fecal samples from each necropsied camel and from other 50 random camels from 3 herds where fatalities occurred, were collected for parasitological examination (flotation method). Toxin analysis was carried out on the intestinal fluids using the mouse test and the MTT (Dimethylthiazol-diphenyltetrazolium bromide test). The pH of the rumenal fluid was immediately measured after the compartment I was opened.

Coccidiosis was confirmed by histology when a severe coccidia infection containing numerous coccidia stages in the intestinal mucosa (more than 1 per high-power filed in several locations) in association with degeneration and desquamation of the intestinal epithelium and a cellurary (eosinophilic) infiltration of the mucosa was seen. In contrast a coccidia-infection was characterized, when only a few coccidia stages and no inflammation of the mucosa as observed. To prove the pathogenicity of *E. cameli*, an infection trial was carried out. Intestinal contents of 25 dissected camels (out of the 106 camels) suffering from coccidiosis were collected and repeated washing and sifting concentrated the oocysts. The oocysts were then sporulated within 6 to 8 weeks in 2.5% potassium diochromate solution under room temperature.

Two, 18 month old camels were artificially infected with 1000-ml solution of sporulated oocysts of E. cameli by the oral route. Over

a period of 6 weeks, fecal samples from both camels were collected every second day and investigated for coccidia oocysts. When one of the infected camels showed intermittent diarrhea, it was euthanized and a detailed post-mortem investigation was performed.

RESULTS

From 106 necropsied, 58 (55%) were infested with coccidia. In group 1 comprising 20 calves no coccidias were found (Table 1), whereas in the other age groups totaling 86 camels, only 28 animals (32.5%) were without coccidias, and 58 camels (67.5%) revealed a coccidial invasion. In 49 (46.7%) camels a coccidiosis and in 9 (8.5%) cases a coccidial infection was diagnosed. The detailed results are summarized in Table 1. Due to the fact that all 20 calves (group 1) were free of coccidia, this group was excluded for further comparison. The main disease in group 1 was either septicemia caused by *Salmonella spp., Streptococcus spp*. and *Staphylococcus aureus* or colonimpation.

Table 1. Distribution of coccidiosis and coccidia-infection in 106 Necropsied dromedaries of different age groups (diagnosed by histopathological investigation).

Group	Number dissected	Age	Coccidiosis	Coccidia infection	No Coccidia
1	20	< 1	0	0	20
2	28	1 - 2	15	2	11
3	40	2 – 12 racing	25	3	12
4	18	> 12 breeding	9	4	5
Total	106	-	49(46.7%)	9(8.5%)	48 (45%)

In all 40 coccidiosis cases of groups 2 and 3, large amounts of barley were seen in the stomach compartments, which resulted in rumen acidosis of compartment I (pH between 5.0 and 6.0). The main pathomorphological findings in these camels were severe hemorrhages (with fresh blood in the intestinal lumen) in combination with a swollen and reddened mucosa of the compartment III, abomasum, jejunum and ileum. In several cases fresh ulcerations were present in the abomasum and the ascending colon was filled with tar-like blood.

Microbiological investigations of necropsied camels of groups 2, 3 and 4 showed that 17.5% suffered simultaneously from a Clostridium perfringens/Bacillus cereus enterotoxemia and coccidiosis (Table 2). The positive mouse test and the positive MTT confirmed these results. In combination with the enterotoxemia the camels developed enlarged lymph nodes and fever above 41 °C.

Table 2. Relations between coccidiosis/coccidia- infection and main diseases in 86 necropsied dromedaries, older than 1 year (Group 2, 3 and 4)

Main diseases	Coccidiosis	Coccidia-Infection	No Coccidia
HD/Clostridiosis (Enterotoxemia)	15 (17.5%)	-	5 (5.8%)
Coccidiosis	23 (26.7%)*	-	-
Ascites, CCN, White Muscle disease	11 (12.8%)	9 (10.5%)	23 (26.7%)
Total: 86	49 (57.0%)**	9 (10.5%)	28 (32.5%)

In 23 of the coccidiosis cases (out of group 2 and 3), no causative agent for enterotoxemia was demonstrated. However, the same pathomorphological findings indicate a simultaneous enterotoxemia as well. Ascites, CCN and white muscle disease were found in the remaining 43 camels (Table 2), 20 out of these camels were also infected by coccidia.

All 49 coccidiosis camels showed a sickness with severe abdominal pain. Fifteen camels developed also bloody feces, but no diarrhea was observed. In combination with the enterotoxemia, ecchymotic hemorrhages of varying severity were also seen subsepicardial as well as subendocardial. All lymph nodes were enlarged, edematous and hemorrhagic. The lungs were congested and showed severe alveolar edema.

In breeding camels (group 4) severe ascites was always found in conjunction with coccidiosis, but no enterotoxemia was observed. In all 49 necropsied camels, which suffered from coccidiosis, numerous coccidia stages (oocysts, macroschizonts and meronts) were located in the mucosa of the jejunum and ileum, but rarely in the duodenum. In 25 camels, where intensive histology on the intestines was carried out (15 locations of the small intestine), the highest concentration of coccidia stages was found in the caudal part of the jejunum and ileum. The size and shape of coccidial stages is presented in Table 3.

Table 3. Description of coccidia stages found in the intestinal mucosa of camels.

Stage	Size (μm)	Shape	Wall	Content
Meront	240 to 330	Round to ovoid	Thin	Numerous merozoites
Macroschizont	240 to 330	Round to ovoid	Thin	Numerous schizoints
Oocyst	Up to 100x80	Piliform	Thick, brown	Non sporulated

Where massive coccidia stages (coccidiosis) were detected, severe edema of the villi, a moderate to severe infiltration of the mucosa with eosinophilic granulocytes and a few macrophages were observed. Where few coccidia stages (coccidia - infection) were found, the intestine showed a mild edema of the mucosa, only. Microscopic examination of lymph nodes and spleen demonstrated a severe sinusedema and in combination with enterotoxemia, an intermediate to severe depletion of the lymphatic tissues, central follicle necrosis and interstitial hemorrhages. Coprostatic examinations of fecal samples from all 106 necropsied camels had negative results for *E. cameli*, but 10 out of 50 fecal samples from three involved herds revealed a few *E. cameli* oocysts.

After repeated washing and sifting of the entire intestinal contents of 25 dissected camels suspected of coccidiosis, the oocysts of *E. cameli* were concentrated and were detected microscopically in all cases. These concentrated oocysts then needed 6 to 8 weeks under room temperature to sporulate. Two young camels, which were artificially infected with these sporulated oocysts by the oral route, excreted the parasite after 6 weeks (prepatency period). Both of the infected camels showed intermittent diarrhea already after 1-week post infection. One of the camels was euthanized and had developed a severe coccidiosis with innumerable, different coccidia stages in the mucosa of jejunum and ileum.

DISCUSSION

Twenty camel calves, 28 young camels, 40 racing camels from three herds and 18 breeding camels totaling 106 camels were investigated for the cause of mortality. Fifty-eight (55%) were diagnosed as having coccidias in their gut, of which 49 (46.7%) revealed a coccidiosis, and 9 (8.5%) a coccidial infection. Forty-eight (45.3%) including all 20 calves were negative for coccidia.

In 15 camels (5 young and 10 racing camels) enterotoxemia caused either by *Cl. perfringens* or *B. cereus* was additionally diagnosed. In 23 camels with coccidiosis, no causative agent of enterotoxemia was demonstrated. However, the same pathomorphological findings in 15 of these camels, indicated a simultaneous enterotoxemia as well. In the remaining cases showing either no coccidias or mild forms of coccidosis (Table 2).

All 49 coccidiosis cases revealed intestinal hemorrhages with massive members of coccidian parasites of different stages. Histopathological investigations showed that at least 5 different locations of the small intestine, especially the caudal ileum and jejunum are required to diagnose coccidiosis. Similar to the first scientific description of camel coccidiosis caused by Eimeria (Globidium) cameli from Henry and Masson (1932) the autors found numerous coccidia stages and a severe infiltration of the mucosa with eosinophilic granulocytes and few macrophages. Three different stages of *E. cameli* (oocysts, macroschizonts and meronts) were identified in the intestinal mucosa (Table 3). It was considered that these stages belong to *Eimeria cameli*, because immature oocysts of *E. cameli* were found within the intestinal epithelial mucosa (Levine, 1985).

It is interesting to note, that camels only older than one year revealed coccidiosis. In young and racing camels more fatal cases were found in conjunction with enterotoxemia and septicemia. The increased fatalities might be explained with special feeding practices for racing purpose in the U.A.E. This is indicated by the detection of an increased amount of barley in the stomach compartments and the associated acidosis, which might have changed the natural intestinal flora. The altered intestinal environment can trigger the growth of *Clostridium perfringens* and *Bacillus cereus* bacteria causing enterotoxemia. As described in chickens (Baba *et al.,* 1997) a concurrent infection with *Eimeria spp*. and *C. perfringens* increases clostridial population in the intestine and has synergic effects on mortality and edema in the upper intestine.

In contrast, breeding camels in the UAE does not receive a high energetic diet and therefore rarely develop enterotoxemia (Wernery *et al.,* 1992). In this group severe ascites was always found in conjunction with coccidiosis.

It is interesting to note that no coccidia were detected in the group of calves. In contrast to other camelids (Haenichen *et al.,* 1994) and numerous domesticated and wild ruminants (Kiefer, 1993; Ziesche, 1994), we found that camel coccidiosis caused by E. cameli is not a disease of calves. This might be due to the fact that fresh alfalfa (the possible source of coccidia) is an important protein source for both young and racing camels, but not for calves (in the UAE). Contamination of fresh alfalfa with coccidia oocysts might occur when alfalfa fields are fertilized with camel or goat manure.

This would then play an important role for the invasion of coccidia oocysts to camels. Another possibility of coccidial invasion is the habit of camels to ingest their own feces. High levels of relative humidity are known to enhance the survival of oocysts outside the host (Stepanova, 1982). The spread of coccidiosis among camels with many reported fatalities during the winter season 1995/1996 in the UAE was most probably associated with a very wet climate.

It is worthwhile to mentioning that *E. cameli* oocysts were rarely found in fecal samples from our camels. Camels from affected herds showed a few *E. cameli* oocysts in only 10 out of 50 tested animals in their fecal samples and routine parasitological investigations of all dissected camels were negative. However, after concentrating the oocysts from the entire intestinal tract we could find in each coccidiosis case the oocysts as well in the feces. This result indicates either a diagnostic problem in our routine parasitological investigation or a low excretion of oocysts from the camel. The simple flotation method might not be adequate enough to isolate the large and heavy oocysts of *E. cameli*. Therefore a combined sedimentation and flotation method may be necessary for routine parasitology. This could also have been the reason, that Hussein *et al.,* (1987) have found *E. dromedarii* being the most prevalent, and *E. cameli* the least in 385 Saudi Arabian camels. Bauer and Buerger, (1984) described the same diagnostic problem with oocysts of *E. leuckartii* in horses, which have the same size as *E. cameli*.

To prove the pathogenicity of *E. cameli*, intestinal contents of 25 dissected camels suffering from coccidiosis were collected and the oocysts were concentrated and sporulated. The oocysts needed 6 to 8 weeks to sporulate. Two young camels artificially infected with sporulated oocysts of *E. cameli* by the oral route excreted the parasite after 6 weeks (prepatency period).

In contrast to other species (cattle, goat and sheep) camel coccidiosis in the UAE is not a disease of the young calves - rather a disease of young camels (older than 1 year). This might be due to the circumstances, that feeding of alfalfa (most probably the source of oocysts) starts in this age group. Camel coccidiosis caused by *E. cameli* plays an important role in intensive camel husbandry in the UAE. Similar finding reported for other species (Baba *et al.,* 1997), a synergism might exist in camels as well between coccidia invasion

and the changed intestinal flora (due to acidosis) developing both enterotoxemia (HD) and coccidiosis.

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