Original Article

Parasitological and histopathological findings of naturally occurring coccidiosis in slaughtered camels (*Camelus dromedarius*) in Aswan Governorate, Egypt

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ABSTRACT

Background: Limited research was conducted on coccidiosis in slaughtered camels in Aswan, Egypt. Accurate screening for camel coccidiosis is critical for an efficient strategic control process.
Objective: To determine the infection rate and histopathological alterations of naturally occurring coccidiosis in slaughtered camels (*Camelus dromedarius*) in Aswan Governorate, Egypt.
Material and Methods: A total of 118 slaughtered camels were included in the study. Specimens from the small intestines were stained with H&E for histopathological examination.
Results: The infection rate of coccidiosis in slaughtered camels was 27.1%. Coccidiosis was more evident in female and adult camels (41.7%, and 42.2%, respectively). Histopathological examination revealed the existence of several stages of *Eimeria* spp. associated with enteritis and accompanied by hyperplasia of gut-associated lymphoid tissue (GALT).
Conclusion: Our results confirmed the high infection rate of coccidiosis in slaughtered camels in Aswan. Several developmental phases of coccidia were detected alongside histopathological lesions within the intestinal tissue.

Keywords: Aswan; camels; coccidiosis; *Eimeria* spp.; histopathology; protozoa; slaughters; Upper Egypt.

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INTRODUCTION

Camelus dromedarius is the predominant species within the *Camelid*ae family. Camels are essential animals in arid regions for centuries due to their ability to tolerate extreme conditions, provide meat and milk, and serve as a means of transportation[1]. There are roughly 35 million camels in the world, mostly domesticated in Africa[2]. Egypt is home to five of the 97 worldwide camel breeds[3,4]. One of the most prevalent global issues affecting the camel population is infection with gastrointestinal parasites[5]. Coccidiosis is considered a parasitic disease of camels attributed to protozoa belonging to the genus *Eimeria*. *Eimeria* spp. are gut-dwelling intracellular protozoa that are transmitted through the fecal-oral route. The non-sporulated *Eimeria* oocysts are released in the feces of infected camels[6,7]. Oocyst sporulation is complete within two to seven days, depending on the coccidian and associated environmental conditions (such as oxygen, temperature, and moisture)[8,9]. Genus *Eimeria* is a monoxenous protozoan. Unlike *T. gondii* that has a broad host range, *Eimeria* spp. are well specialized to parasitize specific intestinal locations in a single host. The life cycle of *Eimeria* spp. comprises an external free-living phase (sporogony) outside the host and a parasitic endogenous phase within the host. In the host, both asexual (schizogony) and sexual (gametogony) reproductive cycles occur, resulting in production of oocysts that are shed into the environment with the feces[9]. There are five recognized *Eimeria* spp. in Old World *camelid*s that infect the camel intestine. Some of these species are quite prevalent among camels and widely distributed. Among them, *E. dromedarii* and *E. cameli* constitute the most ubiquitous and dominant pathogenic species, whereas others (*E. pellerdyi, E. rajasthani*, and *E. bactriani*) are isolated from certain regions with low pathogenicity[6,10].

Coccidiosis in camels is manifested with hemorrhagic enteritis, diarrhea, and dehydration. Both domestic and wild animals can develop subclinical contagious enteritis due to coccidiosis; and in camel calves, it can result in mortality rates as high as 10%[11]. Although previous studies[12–14] revealed that camels were prone to coccidiosis, there are no reports on the infection rate and histopathological changes of coccidiosis in camels in Aswan Governorate, Egypt. Thus, the aim of this study was to determine the infection rate and histopathological findings of naturally occurring coccidiosis in camels in Aswan Governorate, Egypt.

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MATERIAL AND METHODS

This cross-sectional study was conducted at the Department of Pathology, Faculty of Veterinary Medicine, Aswan University, Aswan, Egypt, during the period from January 2024 to May 2025.

Study design: Small intestines of slaughtered camels were microscopically examined. Results were analyzed to calculate infection rate of coccidiosis and determine risk factors.

Study area and sampling: This study was carried out on slaughtered camels for human consumption in different abattoirs in Aswan Governorate, Egypt (24° 5′N latitude, 32° 53′E longitude) during the period from January to September 2024. The total of 118 small intestines of camels of different ages and sexes were collected and examined for coccidiosis. Each specimen was preserved in a separate container in 10% neutral buffered formalin and transported to the laboratory of the Pathology Department, Faculty of Veterinary Medicine, Aswan University, for histopathological and parasitological examination.

Histopathological examination: Promptly preserved specimens were pathologically processed, and 4-5 µm tissue paraffin sections were stained with H & E[15].

Statistical analysis: Data analyses were performed using Minitab v 21. The Chi-square test was applied to determine whether the differences in the infection rate between groups were statistically significant. Statistical significance is considered if $P<0.05$.

Ethical consideration: The study was approved by the Ethical Committee of the Faculty of Veterinary Medicine, Assiut University, Egypt. The registered approved code is 06/2025/0309.

RESULTS

The infection rate of coccidiosis in slaughtered camels was estimated at 32/118 (27.1%), affecting 41.7% and 20.77% of females and males, respectively, 42.2% and 17.8% in adults and young, respectively. The infection rate was significantly higher (P=0.004.) in adult camels (>4 years) compared to young ones (<4 years). Similarly, the infection rate was significantly (P=0.019) more common in females than males. (Table 1).

Table 1. The prevalence rate of coccidiosis in slaughtered camels.

Variable	Not infected (%)	Infected (%)	Statistical analysis
Age			
>4 years (adult)	26 (57.8)	19 (42.2)	$\chi 2 = 8.4$
<4 years (young)	60 (82.2)	13 (17.8)	$P = 0.004$
Sex			
Male	65 (79.3)	17 (20.7)	$\chi 2 = 5.54$
Female	21 (58.3)	15 (41.7)	$P = 0.019$
Total	86 (72.9)	32 (27.1)	

Microscopic examination of histopathological sections revealed several stages of *Eimeria* spp. (macrogamonts, microgamonts, schizonts, and oocysts) in the mucosal layer between the intestinal glands in the lamina propria. Parasitic stages were associated with necrotizing enteritis, collapse of the affected villi and crypts, hyperplasia of GALT (Fig. 1A), and significant infiltration of inflammatory cells, mostly lymphocytes and macrophages (Fig. 1 A-F). Additionally, there was glandular compression atrophy (Fig. 1 B, D, F) and in-between edematous effusion (Fig. 1 C-E). The epithelium of the intestinal glands showed irregularity and crowding (Fig. 1 B-F), and necrosis (Fig. 1 C-D).

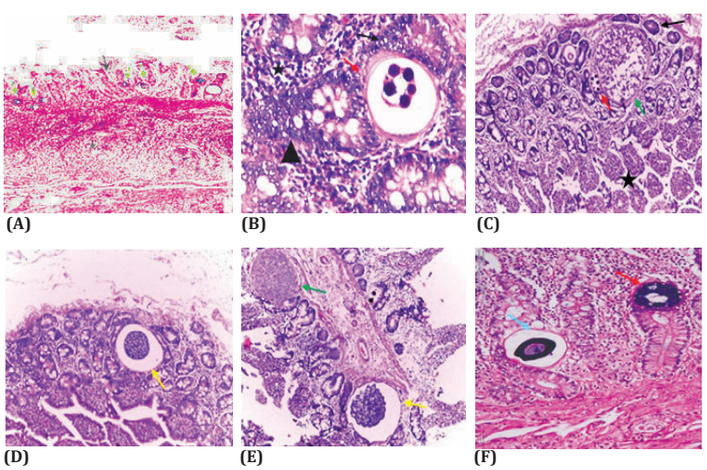

Fig. 1. Microphotographs of camel small intestines showing: **(A)** Different stages of *Eimeria* spp. in the mucosal layer between the intestinal glands in the lamina propria (green arrows) and severe necrotizing enteritis with collapse of the affected villi and crypts (black arrow). Hyperplasia of GALT (star) was observed at ×20; **(B)** Macrogamont (red arrow) in the lamina propria with distinct, varying-sized wall-forming bodies at the periphery of cytoplasm associated with compression atrophy of intestinal glands (black arrow). The epithelium of the intestinal glands shows irregularity and crowding (head arrow) with infiltration of mononuclear inflammatory cells (star) between the intestinal glands ×100; **(C)** The developing microgamont (green arrow) between the intestinal glands in the lamina propria contains multiple irregularly arranged, highly condensed nuclei with blastophore formation. Adjacent is a developing macrogmont (red arrow). Necrosis of the epithelium of intestinal glands (black arrow) and edema (star) were noted x40; **(D)** Schizont (yellow arrow) containing numerous merozoites is surrounded by a prominent parasitophorous vacuole x40; **(E)** Schizont (yellow arrow) and microgamont (green arrow), x40; **(F)** The whole interior of *Eimeria* spp. oocyst (blue arrow) is occupied by a centrally-nucleated sporont, and the oocyst possesses a micropylar cap. A macrogamont (red arrow) is at the base of intestinal villi x40.

DISCUSSION

Coccidiosis is a major parasitic cause of neonatal diarrhea in livestock, particularly camels[16,17]. The results of the current study revealed a high rate of coccidiosis among camels in Aswan abattoirs, southern Egypt. Our recorded overall infection rate of coccidiosis in 118 camels (27.1%), was closely related to the Abbas *et al*.[18] report of 38% in slaughtered camels in Cairo, Egypt, and the 30% detected in camels in Saudi Arabia[19]. However, our present record was higher compared to that reported by Djerbouh *et al*.[20], who confirmed coccidiosis in 9.6% of Algerian camels. Our result was lower than those reported in Algeria (45.93%)[11], and Turkestan (42.5%)[21]. These varying infection rates are probably a result of overall disparities in geographical distribution as well as the impact of variations in environmental factors on the sporulation of *Eimeria* spp. oocysts[21]. Being monoxenous parasites, the *Eimeria* spp. life cycle is composed of a free-living sporogony phase in the external environment, and a parasitic endogenous phase inside the host[22]. Jn the host, the parasite undergoes asexual merogony and sexual sporogony cycles that produce the immature oocysts released in the enviroment[8].

According to the current findings, the infection rate of coccidiosis was greater in females (41.7%) than in males (20.7%). This outcome is consistent with several studies[11,23,24] in which the investigators indicated that coccidiosis was more common in females than in males. Females may be more susceptible to infection because of physiological traits that predispose to lower their immunity to diseases and function as stressors. Furthermore, older camels had a greater frequency of coccidiosis (42.2%) compared to younger animals (17.8%) which agrees with the report by Boukert *et al*.[11]. This may be attributed to the aging of the intestinal mucosa and lack of elasticity. In addition, reduction in the number of intestinal villi in aged animals can reduce absorption, and fewer intestinal cells may lead to decreased immunological effectiveness.

Our histopathology findings demonstrated necrotizing and lymphocytic enteritis in naturally infected one-humped camels (*Camelus dromedarius*) with various *Eimeria* spp. developmental stages, such as gamonts, schizonts, and oocysts, in the mucosal layer between the intestinal glands in the lamina propria. These results agree with the findings observed by Boukert *et al*.[11] who observed a significant incidence of duodenal epithelial desquamation and enteritis with various developmental stages of *Eimeria* spp in camels. Hemorrhages on the mucosal surface and intestinal epithelial erosion, especially in the crypts, were observed indicating gross lesions. In this context. *E. cameli* infections are widespread in camels and can lead to enteritis[25–27]. Moreover, camels in India were shown to harbor schizonts, gamonts, and oocysts in their duodenum and cecum[27]. Furthermore, our findings align with previous studies[28-30] that documented comparable histopathology findings in the intestines of birds infected by *Eimeria* species. In many animals, *Eimeria* spp. are the primary intestinal tract pathogens, infiltrating and causing damage to the intestinal epithelium[31]. According to Dubey *et al*.[6], *E. cameli* was detected in all intestinal locations, including the lamina propria and submucosa, especially in the Lieberkuhn crypts. Sazmand and Joachim[26] identified eosinophilic enteritis and large schizonts in the lamina propria and Lieberkuhn gland epithelium.

The intestinal lining becomes infected with *Eimeria* spp. stages causing tissue damage, poor nutritional absorption, blood loss, dehydration, and decreased feed intake[32,33]. Sporozoites first penetrate surface enterocytes before entering intraepithelial lymphocytes. Following their departure from the epithelium, these lymphocytes pass through the lamina propria and enter the crypts. As the schizonts develop, merozoites are released, and they have the ability to enter new intestinal cells and produce new generations. The schizonts may rupture, causing damage to the intestinal wall, severe internal bleeding, and reduced absorption ability[34].

Coccidiosis-induced cellular damage and compromised immune responses provide an environment that other infections can exploit to inflict more harm. When *Eimeria* parasites invade and rupture the intestinal epithelial cells, the protein concentration in the intestinal lumen rises, creating an optimal environment for the proliferation of pre-existing *Clostridium perfringens* and the onset of secondary necrotic enteritis[35]. *Eimeria* spp. causes alterations in the intestinal mucosa, which can progress to various intestinal diseases. One primary example is the proliferation of *C. perfringens*, which causes necrotic enteritis, as well as *Salmonella, E. coli,* and *Campylobacter*[36]. Furthermore, cell-mediated immunity plays a crucial role in infection resistance. The T-lymphocytes react to coccidial infections by generating cytokines and attacking infected cells directly[37]. The predominance of *Eimeria* spp. and their resistance to therapy exacerbates the situation significantly[38].

In conclusion, this research has yielded important information about the infection rate and histopathological findings of coccidiosis in slaughtered camels in Aswan Governorate. It is advised that further epidemiological research be done to address coccidiosis in the dromedary camel population in Egypt through preventive and control measures.

Author contribution: Ahmed M and Abdelhamid M proposed the study topic and design and performed the practical examination. Ahmed M, Bakr L, and Abdelhamid M described the results, wrote the draft, and finalized the article for publication. All authors

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