

CONCURRENT INFECTION OF COCCIDIOSIS AND HAEMONCHOSIS IN A DROMEDARY CAMEL CALF FROM RAJASTHAN, INDIA

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Haemonchosis is considered as the most severe gastrointestinal helminthosis of camels which may be associated with clinical disease and can be fatal. This nematode occurs in the abomasum of the infected camels sucking blood from the mucosal vessels leading to haemorrhagic anaemia, a characteristic feature of the disease (EL Hassan *et al*, 2011). Likewise, coccidiosis may be seen in camel calves with symptoms like diarrhoea, dysentery, dehydration, rough hair coat and anaemia (Parsani *et al*, 2008). Among gastrointestinal protozoan parasites, infection of *Eimeria* spp. is a major problem in camels. The present study is the first description of a case where in the pathology of concurrent coccidiosis and hemonchosis infection has been studied in a dromedary camel calf.

Case History and Examination

A one year old dead dromedary camel calf of an organised camel herd with a past history of prolonged emaciation, progressive anaemia, anorexia, debility and intermittent diarrhoea during winter was presented for postmortem examination. The calf was under treatment with supportive drugs and intravenous fluid but its condition did not improve and it succumbed. The necropsy was performed immediately after its death and gross lesions were observed. Tissue samples from major organs were collected and fixed in 10% formal saline for histopathology. The small and large intestinal contents were also collected in sterile vials for parasitological examination using floatation technique and direct microscopy.

Results

The gross examination of the camel calf revealed severe gelatinisation and atrophy of subcutaneous and visceral fat and pale mucous membranes. The thoracic and abdominal cavity contained moderate amount of clear watery fluid. The heart has litchi like appearance

due to severe atrophy and gelatinisation of epicardial fat (Fig 1A). The other important gross changes were observed in gastrointestinal tract. The abomasum was found empty and the mucosa showed thickening, congestion and multiple petechial haemorrhages (Fig 1B). The small intestinal mucosa also showed thickening with marked corrugation and presence of yellowish watery mucous mixed contents (Fig 1C). The mesenteric fat was atrophied, gelatinised and showed prominent blood and lymphatic vessels (Fig 1D). The mesenteric lymph nodes were pale, enlarged and oedematous (Fig 1D). The lung was pale pink in colour and showed mild emphysema. No significant gross changes were observed in other organs.

The small intestinal contents on microscopic examination revealed presence of numerous oocysts of *Eimeria* spp whereas, the large intestinal contents revealed the eggs of *Haemonchus* spp. The eggs were thin-shelled, oval shape with equal poles and morula not fully filled the shells of the eggs (Fig 2A). The oocysts of *Eimeria cameli* were oval shape, distinctly brown coloured, had a single large sporont with the presence of micropyle on the 'pointed' end (Fig 2B).

The histopathology of abomasal mucosa showed moderate infiltration of eosinophils and mononuclear cells. In addition, haemorrhages, congestion of blood vessels in lamina propria and desquamation in the apical border of the villi was observed in the abomasum. The histopathological examination of small intestine showed presence of large number of different developmental stages of *Eimeria cameli* inside the mucosal layer (Fig 3) which caused severe degeneration and desquamation of the intestinal epithelium. The affected villi and crypts were also distended and disorganised due to presence of developmental stages such as giant schizonts, microgamonts, macrogametocytes and oocysts of *E. cameli*. The intestinal mucosa showed mild to

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moderate mononuclear and eosinophilic infiltration in the lamina propria (Fig 4). The cortical area of mesenteric lymph node also showed occasional presence of developmental stages of *E. cameli* along with eosinophilic infiltration (Fig 5). The important histopathological lesions observed in other organs were mild necrotic and degenerative changes in heart and mild emphysema in lung.

Discussion

The gastrointestinal parasites play a significant role in camel husbandry because parasites not only reduce the productivity and performance of camels but also predispose them to other infectious diseases (Radfar and Gowhari, 2013). Due to the common clinical signs and acute nature of gastro-intestinal helminths, it is practically impossible to distinguish these diseases. The general clinical signs shown by camel calf of the present case are frequently

reported in most of the gastrointestinal parasitic infections including haemonchosis (Arzoun *et al*, 1984; Saminathan *et al*, 2015) and coccidiosis (Kinne and Wernery, 1998; Kumar *et al*, 2015).

The gross pathological changes of fluid in the body cavities due to hypoproteinemia, atrophied and gelatinised subcutaneous and visceral fat and petechial haemorrhages in abomasal mucosa as recorded in the present case, corresponded with the haemonchosis infection in sheep (Saminathan *et al*, 2015). The gross changes in small intestine were also more or less similar to *E. cameli* infection in camels reported in a previous study (Kumar *et al*, 2015). The histopathological changes of eosinophilic and mononuclear cell infiltration observed in the abomasum of affected camel due to haemonchosis is similar to that reported in sheep (Saminathan *et al*, 2015). Likewise, the histopathological changes in intestinal mucosa due to presence of different

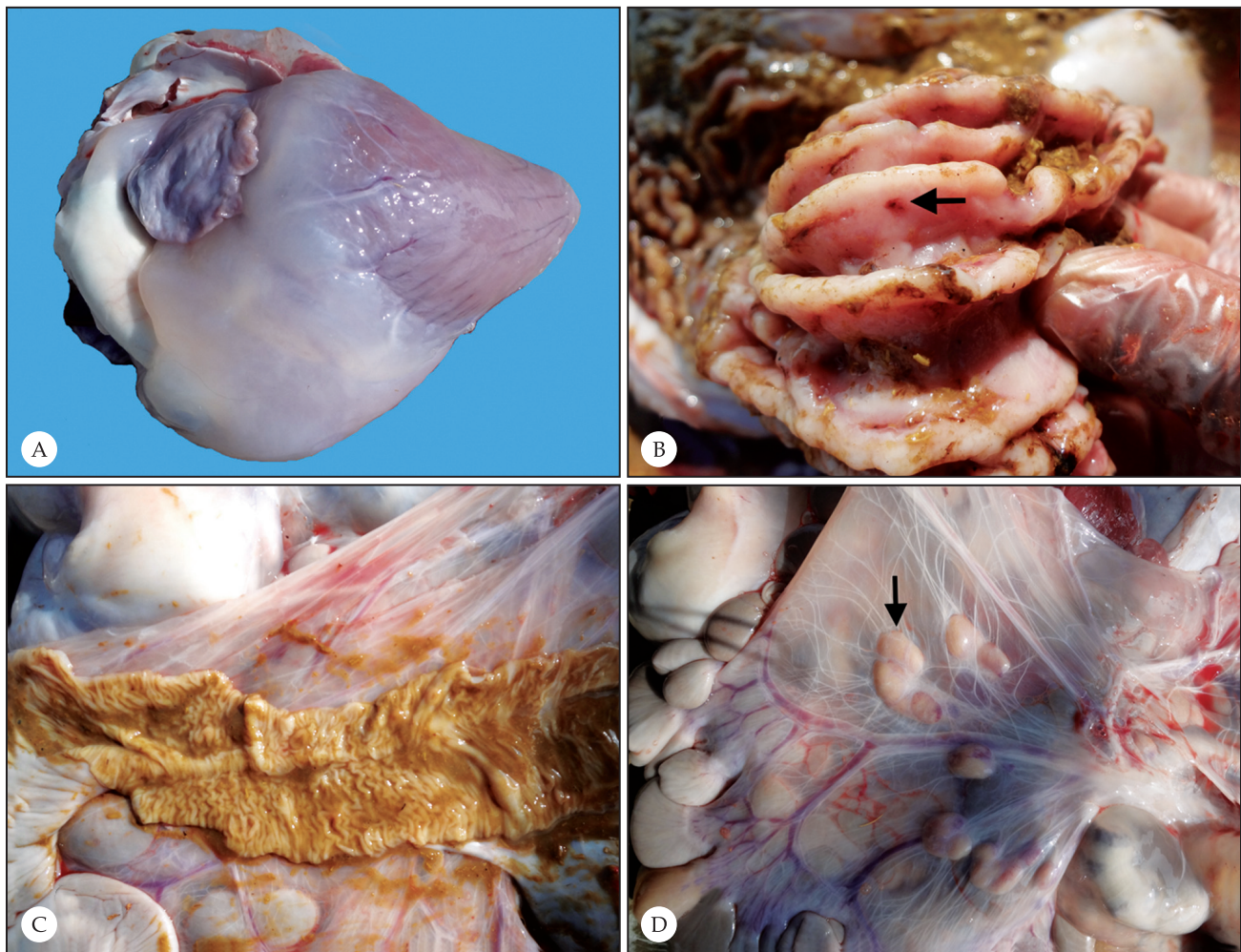


Fig 1. A. Litchi like appearance of heart due to gelatinisation of epicardial fat, B. Abomasal mucosa showing thickening and petechial haemorrhages (arrow), C. Mucosa of small intestine showing thickening, corrugation and presence of watery mucous mixed contents, D. Enlarged mesenteric lymph nodes (arrow) with prominent blood and lymphatic vessels along the mesentery.

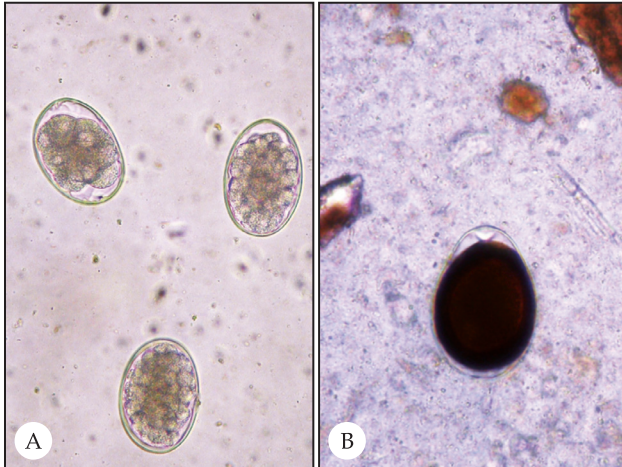


Fig 2. A. Eggs of *Haemonchus* from intestinal contents, B. An oocyst of *Eimeria cameli* from intestinal contents.

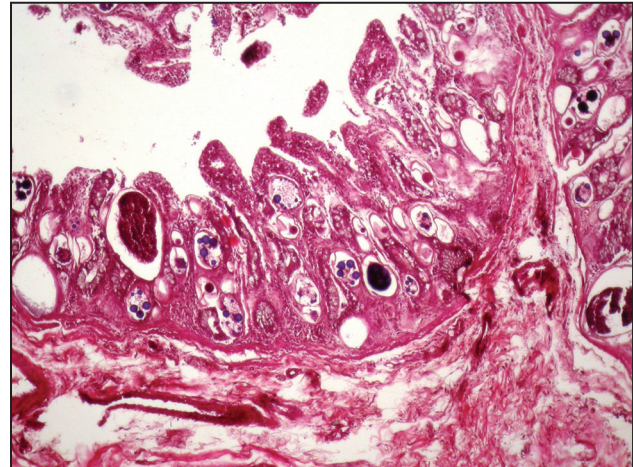


Fig 3. Various developmental stages of *Eimeria cameli* in the mucosa of small intestine (H & E X100).

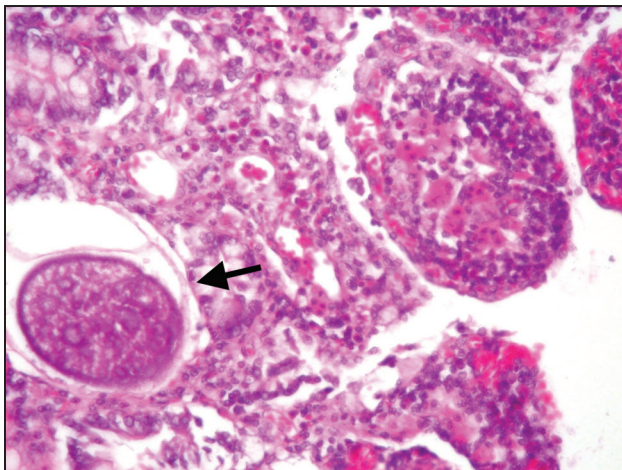


Fig 4. A large schizont of *Eimeria cameli* (arrow) containing merozoites surrounded by infiltration of eosinophils and mononuclear cells in the mucosa of small intestine (H & E X400).

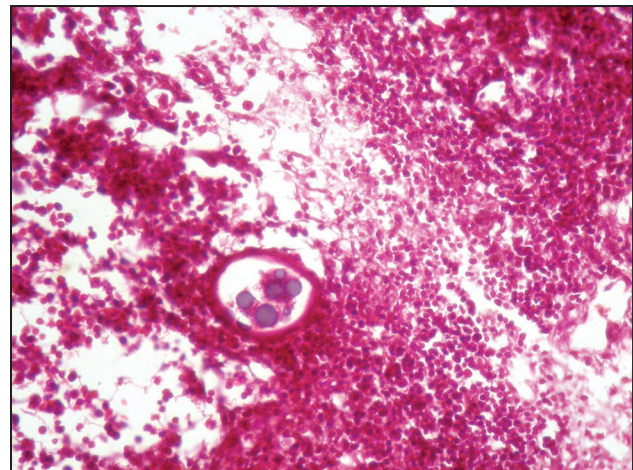


Fig 5. Mesenteric lymph node showing presence of oocyst of *E. cameli* along with eosinophilic infiltration (H & E X400).

developmental stages (oocysts, large schizonts, meronts and macrogamonts) of *E. cameli* infection were comparable with those described in previous studies in camels (Kinne and Wernery, 1998; Kumar *et al*, 2015). The coccidian parasites were also observed in the mesenteric lymph nodes of the present case which is contrary to the finding in sheep and goats (Tafti and Mansourian, 2008).

The differences between *Eimeria* spp. and their prevalence depend on some factors such as environment, animal factors, farm management and other factors (illness and stress) but clinical manifestations appear mostly under stressful conditions (Yakhchali and Cheraghi, 2007). The camel calf of the present study was also thought to be under stress due to concurrent *H. longistipes* infection and this may be the one of the reasons for more severe

clinical signs and pathological lesions. In agreement with the finding of the present study, a significant correlation between age and severity of coccidiosis was observed in previous studies. The camel calves were found to be more infected with the *Eimeria* spp. than older ones, while adult camels were found to be chronic shedders of oocysts without manifesting clinical signs (Radfar and Gowhari, 2013). Contrary to this, camels only older than one year and adults revealed *Eimeria* coccidiosis in some studies (Kinne and Wernery, 1998; Sazmand *et al*, 2012). This might be due to the contamination of grazing pasture with coccidia oocysts by other camels. Another possibility of coccidial invasion is the habit of camels to ingest their own faeces (Kinne and Wernery, 1998). The winter season may also enhance the survival of coccidian parasite and the rate of infection was found higher in the winter season (Kinne and Wernery, 1998; Sazmand *et al*, 2012). The infected camel calf of

the present case also died during peak winter season. Besides this, one of the important factors was the immunosuppression due to *Haemonchus* infection. The fatal enteritis in camels caused by synergism between coccidiosis and haemonchosis has been reported in a previous study (Iyer *et al*, 1968). *H. longistipes* has been predominantly detected from the camels of Rajasthan region (Gahlot and Chhabra, 2009). The morphology of eggs and cultured larva of *Haemonchus* in the present study also corresponded with the *H. longistipes*.

On the basis of observations of the present study, it was concluded that the possible pathogenic mechanism responsible for cause of death in the camel calf of the present case having concurrent haemonchosis and coccidiosis infection could be haemorrhagic anaemia, hypoproteinemia and diarrhoea causing fluid loss and dehydration leading to hypovolemic shock and severe pathological consequences.

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