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MÉMOIRES ORIGINAUX

An outbreak of haemorrhagic gastro-enteritis in camels *(Camelus dromedarius)*

EPIDÉMIE DE GASTROENTÉRITE HÉMORRAGIQUE CHEZ LES CHAMEAUX (*Camelus dromedarius*)

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Résumé

Une gastro-entérite catarrhale hémorragique, accompagnée de diarrhée sanglante et de douleurs abdominales, a été constatée chez des chameaux du Punjab oriental, aux Indes.

La présence dans l'intestin de stades évolutifs d'*Eimeria* (*Globidium*) *cameli* et d'*Haemonchus longistipes* a conduit les auteurs à rechercher le rôle éventuel du premier de ces parasites dans la symptomatologie ci-dessus évoquée, et la part qui pourrait lui être attribuée dans la constitution des lésions histopathologiques, observées dans le tube digestif.

Summary

A catarrhal, haemorrhagic gastro-enteritis in camels accompanied by blood tinged diarrhoea and abdominal pain and associated probably with the developmental stages of protozoan parasite *Eimeria* (*Globidium*) *cameli* and *Haemonchus longistipes* is described. The possible pathogenic role of the former parasite is discussed on the basis of histopathological findings.

Introduction

An investigation into the causes of certain unexplained mortalities in camels in East Punjab (India) fortuitously brought to light the existence of a haemorrhagic gastro-enteritis in camels associated with *Eimeria* (*Globidium*) *cameli,* Henry and Masson, 1932, and *Haemonchus longistipes,* Railliet and Henry, 1909, infection in this country. Results of the study of the pathology and parasitology of the disease are communicated in this paper. The literature on globidiosis has been reviewed by Pols (1960) who has also discussed the taxonomic status of the parasite. The present authors have followed Reichenow's (1953) description and diagnosis of the endogenous stages of the parasite, *Eimeria* (*Globidium*) *cameli.*

Materials and methods

Sudden deaths in camels were first observed in 1959 and the symptoms as reported by the camel owners were abdominal pain and a blood-tinged diarrhoea, but the condition was overlooked. Two deaths in a population of 120 camels occurred in a village near Faridkot, in May 1960. In June-July 1960, the disease spread to 18 villages in Faridkot, Muktsar and Ferozepore districts affecting about 1200 camels and causing one to 40 percent deaths. About two to three percent of the affected animals recovered. The disease had practically subsided at the time in September 1960 when we were called upon to investigate. Only one ailing animal was available for study, and necropsy was carried out on this as well as two other dead camels. The latter had been dead for about 8 and 16 hours respectively.

Morbid tissues which were collected from two already dead camels and one destroyed 'in extremis' formed the material for the studies. Heart blood and intestinal contents were cultured for bacteria and fungi on different aerobic and anaerobic media.

Blood from the ailing animal, and filtered and unfiltered suspensions of spleen from this as well as two other dead camels were inoculated into batches of guinea-pigs, rabbits and mice, subcutaneously and intra-peritoneally. The suspensions were also inoculated into susceptible hill bulls to exclude rinderpest. Filtrates of intestinal contents were tested for toxicity in mice. Microscopic examination of blood smears and faecal samples from these cases, from a few of the incontact and apparently healthy camels, and also from a few recovered camels was carried out. Transmission with morbid materials to other camels was not attempted.

Results

A. **Symptoms :** Two principal forms of the disease, the acute and chronic types, were recognised by the local Veterinary Investigation staff. There were a few peracute cases too, in which some animals collapsed and died suddenly while at work without manifesting any symptoms. The acute form was characterised by malaise, listlessness,

anorexia, rigors, incoordination of movement, tucking in of the abdomen, kicking at the belly frequently, and muco-sanguineous stringy faeces accompanied by marked tenesmus. In the terminal stages, there was cold sweating, dyspnoea, hypothermia, weakness of hind quarters and prostration. Death supervened usually in 4 to 12 hours following the onset of the symptoms. In chronic cases, the symptoms were restlessness, frequent attempts to squat on cold surfaces, and general debility. The faeces were, to begin with, semi-solid or soft, with a tinge of mucus and blood, and gradually assumed a dark charcoal hue. In the later stages, diarrhoea was pronounced, the faeces containing blood clots, bile, and mucus in varying amounts. Treatment of the affected animals by antibiotics was infructuous, but some animals, which were sick and had voided dark coloured faeces for about a week, made an uneventful recovery.

The disease was confined to the dromedary. although cattle, buffaloes, shepp, goats, and poultry were kept in close contact with the affected animals.

In the affected villages, a number of camels were unthrifty and had depraved appetite suggestive of mineral deficiency/deficiencies.

The main feed of the animals consisted of the leaves of the Cluster Bean (*Cyamopsis psoraliodes*). The feed could not, however, be incriminated as in a majority of outbreaks only one animal or two in a herd of 10-20 was reported to have been lost.

B. **Bacteriological and virological examination :** Cultures from heart blood and intestinal contents yielded no organisms of pathogenic significance. In one instance, intestinal clostridia were recovered which were considered of no importance as the filtrates caused no ill effects in mice.

The biological test helped to exclude rinderpest. The rabbits, guinea-pigs and mice which received the infective suspensions remained unaffected for a period of three weeks observed.

C. **Pathology :** The ailing camel revealed marked anaemia and hydraemia. The total erythrocyte and leucocyte counts were 1.56 millions and 13,2 thousand per c.mm. respectively and the differential leucocytic count revealed marked eosinophilia (14 %). In the incontacts and the recovered. the number of eosinophiles ranged from 6 to 21 percent.

Gross pathology : The carcases were poor in condition with marked depletion of body fat.

The abomasal contents were semi-solid, containing considerable amounts of mucoid-exudate and blood clots. The mucosa of the pyloric antrum was hyperaemic and covered by a patchy layer of adherent muco-sanguineous exudate.

The small intestine, particularly the duodenum, was severely inflamed, the lumen containing dark coloured clotted blood and mucus admixed with the ingesta. The exudate at places was firmly adherent to the mucosa and its dislodgement caused peeling of the epithelium in shreds. The colon and caecum were moderately congested, the lumina containing semi-solid faecal balls coated with blood and mucus.

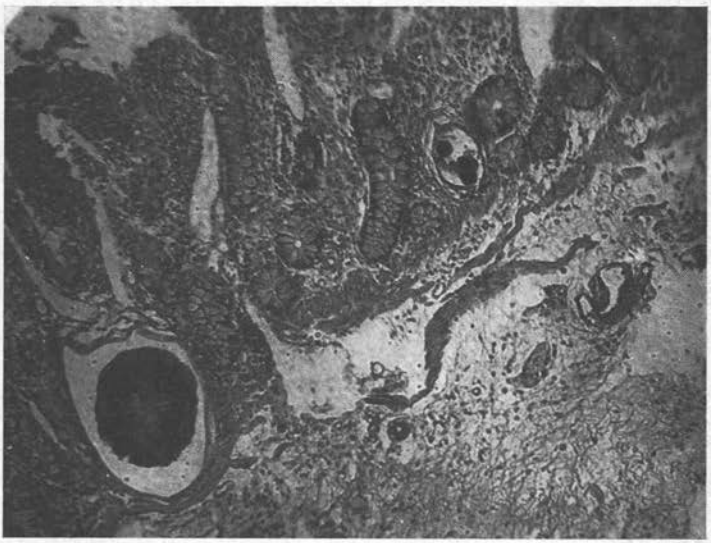

FIG. 1. — Section of duodenum showing a schizont and macrogametocyte in the Brunner's glands. Note the general disorganisation of the mucosa and thickening of villi due to cellular infiltration. H. & E. X. 120

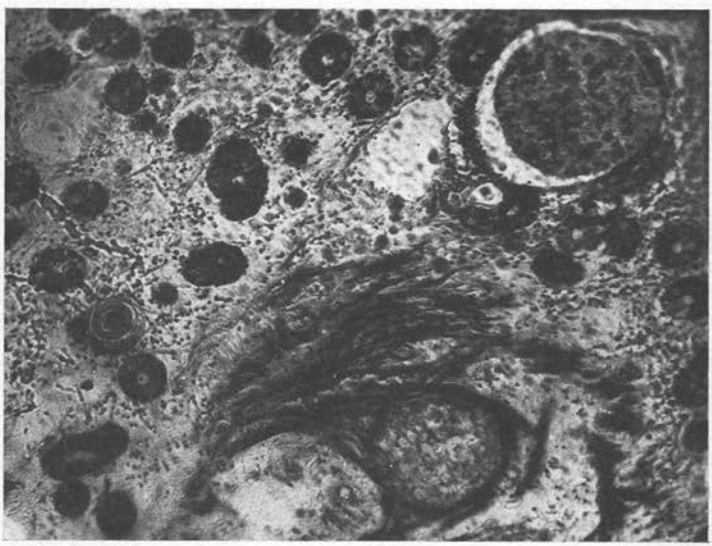

FIG. 2. — Section of duodenum showing development of schizonts in the Brunner's gland and muscularis mucosa and the cross sections of an unripe oocyst on the left, H. & E. X. 120

The spleen was pale and its pulp considerably reduced.

Histopathology : The overall microscopic picture was one of acute and subacute, catarrhal gastro-enteritis, with occasional focal haemorrhages. In the abomasum, there was varying degree of desquamation of the surface epithelium in the pylorus, obliteration of gastric pits with muco-sanguineous exudate, and regressive changes in the epithelial cells of the gastric glands. There was marked infiltration of the lamina propria and subepithelial layer with lymphocytes, plasma cells, stray eosinophiles, neutrophiles and macrophages. There was widespread capillary hyperaemia in these

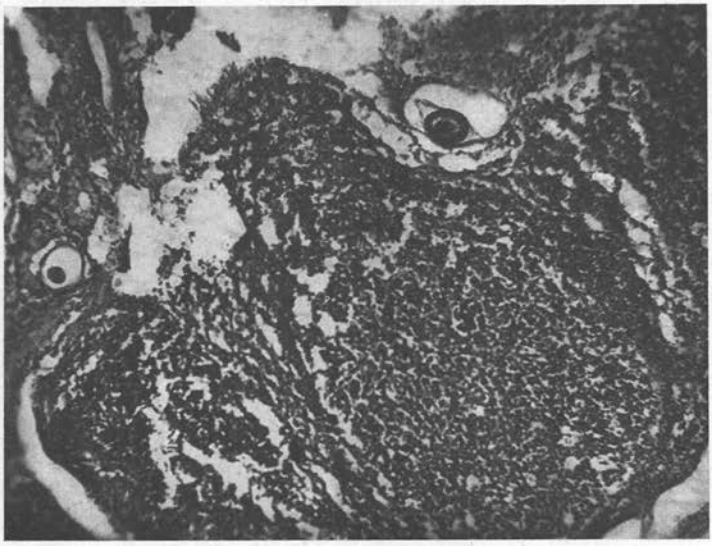

FIG. 3. — Section of cæcum showing hyperplasia of lymph follicles. Note the disorganisation of overlying lamina propria and the cross section of an unripe oocyst on the right, H. & E. X. 120

two layers. At places, a tenacious muco-sanguineous exudate was found adherent to the surface epithelium and/or filling the gastric foleolae.

In the duodenum, the villi were greatly distended and disorganized due to marked cellular infiltration. Most of the villi were bereft of the lining epithelium and their borders were uneven. There was varying degree of capillary congestion and oedema. The infiltrating cells were lymphocytes, plasma cells, eosinophiles and macrophages. A slightly less marked cellular infiltration, mainly perivascular. was also observed in the subepithelial layer. The Brunner's glands revealed slight disorganization of structures, hyper-secretion and/or necrobiotic changes in the epithelial cells due to infestation with the developmental stages of the parasite, *Eimeria* (*Globidium*) *cameli* (fig. 1). The pasarites were not observed in the surface epithelial cells. A few parasitic cysts were occasionally seen lodged in the muscularis mucosa (fig. 2). There were also a few degenerate cysts infiltrated by neutrophiles and macrophages.

In the caecum, the surface epithelium exhibited degenerative changes and a variable, but often considerable, degree of desquamation into the lumen. The cellular infiltration of the lamina propria and the subepithelial layer was similar to that observed in the duodenum. The lymph follicles were markedly hyperplastic. A few developmental stages of the parasite were seen embedded in the mucosa at varying depths, but principally at the basal level of the glands (fig. 3).

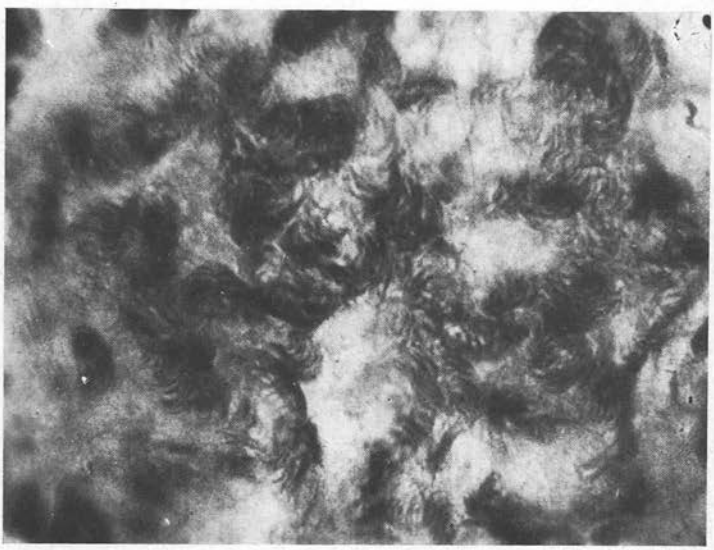

FIG. 4. — Mature schizont showing whorls like arrangement of Merozoites, H. & E. X. 960

D. **Parasitological findings :** Faecal samples showed that almost every camel examined was heavily infested with nematode eggs, the average count ranging from 100 to 16,000 per gram of faeces. The three camels necropsied gave a count of 9000, 15,000 and 16,000 eggs respectively. Mature specimens of *Haemonchus longistipes* were recovered from the abomasum and duodenum in large numbers in these cases. The camels which survived the disease gave a count varying from 100 to 800 eggs per gram of faeces.

Endogenous stages of *Eimeria* (*Globidium*) *cameli* recognized were schizonts, macro- and microgametocytes which were diagnosed on the basis of descriptions given by Enigk (1934) and Reichenow (1953).

Schizonts in various stages of maturation were seen. Under low magnification they appeared as 'cysts' embedded in the mucosae. Mature schizonts measured 500 × 900 μ and contained numerous minute crescent-shaped merozoites arranged in whorls arising from the margin of protoplasmic spheres in the stroma of schizonts (fig. 4).

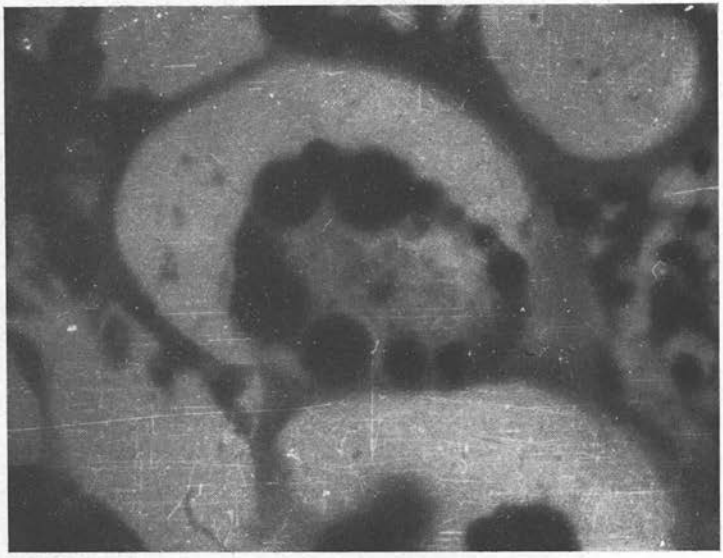

FIG. 5. — Oil immersion view to show the presence of microgametes, H. & E. X. 1100

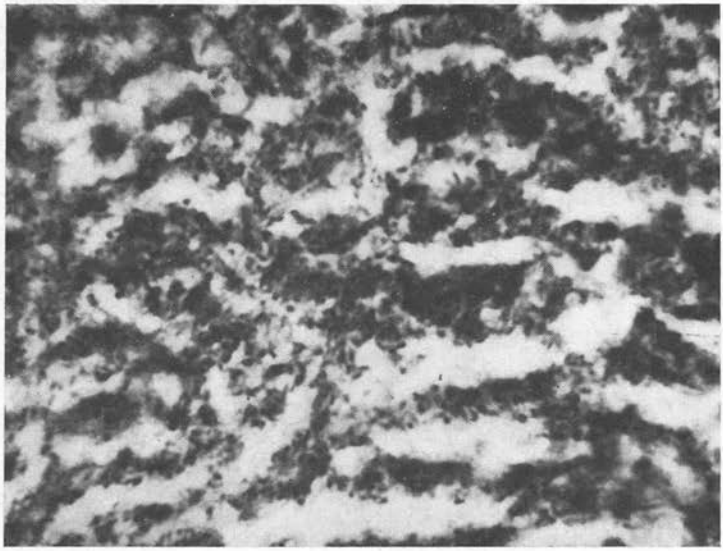

FIG. 6. — A stage in the maturation of macrogametocyte. Note the peripheral arrangement of globidial bodies close to the cell wall, the nucleus and eccentric karyosome, H. & E. X. 960

In fresh tease preparations, macrogamètes measured 102 × 110 μ. The macrogametocytes contained a small central nucleolus and a number of eosinophilic globules arranged peripherally, in the form of a ring close to the inner wall of the cyst (fig. 5).

The mature microgametocytes were almost similar in morphology to the schizonts except that the cysts were smaller in size and contained numerous microgametes which appeared as stumpy rods (fig. 6).

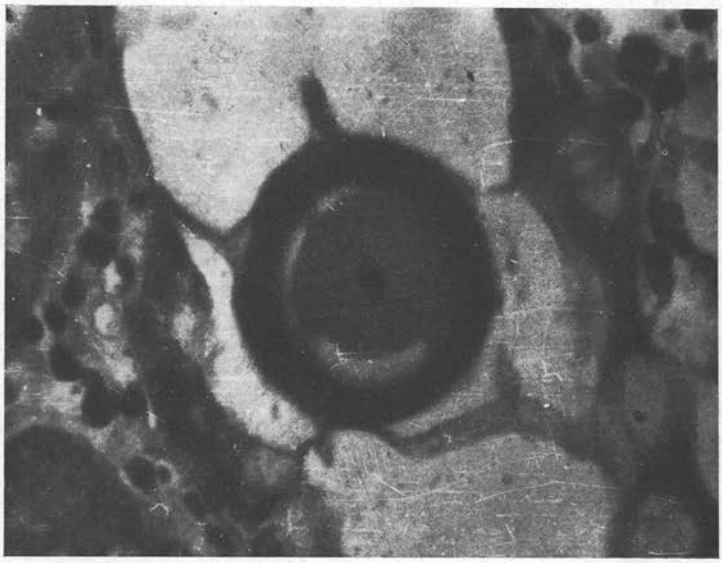

FIG. 7. — Cross section of an unripe oocyst. Note the double layered outer wall and the unsegmented sporont within, H. & E. X. 960

The oocysts conformed to the description of Henry and Masson (*loc. cit.*). They were horse-shoe shaped in sections but ovoid in teased preparations, yellowish brown, 80 × 100 μ × 60-70 μ in size, and double layered (fig. 8). The outer layer was finely granular, 13-15 μ thick and the inner layer thin and translucent. The micropyle measured about 27 μ with a distinct polar cap, the height of which being 13 μ. The sporont was oval and granular and measured 66 × 48 μ. Small elliptical structures apparently corresponding to trophozoites and containing a pair of globular bodies were detected in the sporont.

The different endogenous stages detected were located in the epithelial cells of Brunner's glands in the duodenum and to a lesser extent in the mucosa of the caecum. Portions of the jejunum and ileum were unfortunately not collected.

Discussion

The existence of the disease condition characterized chiefly by varying degree of gastro-enteritis and associated with the presence of cystic stages of the parasite

Globidium has been reported in domestic animals from time to time, and the reports of Marsh and Tunnicliff (1941), and Rac and Wilson (1959) justify the suspicion regarding the pathogenic role of the parasite. In experimental infections due to *Eimeria bovis* — a parasite which is held by several workers, notably Hammond *et al.* (1946), to be closely similar to *Globidium smithi pillers* (1928), in respect of the morphological characteristics of the developmental stages — the symptoms and histopathological changes were demonstrated to be associated with the development of gamegonous stages of the parasite. Diarrhoea, usually commencing about the time the first oocysts were discharged, was seen to be followed, in severe infections, by dysentery accompanied by tenesmus. In the absence of transmission trials with mature

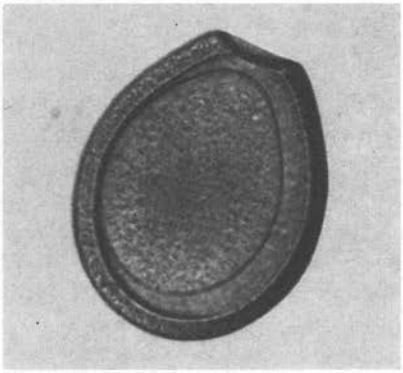

FIG. 8. — An unripe oocyst from a potassium hydroxide digest preparation of duodenal mucosa. Note the micropyle, outer and inner walls and the unsegmented sporont, H. & E. X. 540

oocysts in camels, or in other susceptible experimental animals, it is difficult attribute this gastro-intestinal syndrome in camels to the developmental stages of *Eimeria* (*Globidium*) *cameli*.

The presence of haemorrhage in the abomasum, duodenum and caecum appeared to be far in excess of the degree of microscopic changes observed in these organs. Vascular phenomena were, microscopically, no more intense than wide-spread capillary hyperaemia and mild oedema. The chances of capillary haemorrhages occurring in the course of desquamation of the surface epithelium and also as a result of depredations of *Haemonchus longistipes* cannot, however, be ignored. The severe anaemia and oligocythaemia observed are perhaps attributable to the haemorrhages incident to the heavy helminthic infection. It is pertinent to mention in this connection that the regional investigation staff had observed that only the small intestine was the seat of intense inflammatory changes and haemorrhages in the cases necropsied by them — a finding which would raise the doubt whether *Haemonchus longistipes per se* could have brought about the mortality in camels. However, it is not unlikely that under conditions of stress associated with helminthiasis and single or multiple mineral deficiencies the parasites ordinarily held to be non-pathogenic like *Globidium* could propagate itself unchecked causing morbidity and mortality in camels.

Neither bacteria nor their toxins appear to be responsible for the haemorrhagic

syndrome in camels. The lack of thermal reaction in these animals, absence of morbidity in the different laboratory animals which received the filtrates of spleen and blood, and also the absence of significant cellular changes in the organs such as liver, spleen, lymph nodes and the brain would eliminate the possibility of a virus as being involved. The authors are aware that if transmission trials in camels had been conducted the value of these observations would have been enhanced.

The assumption that the parasite described is *Eimeria* (*Globidium*) *cameli* finds support in the demonstration of the different characteristic endogenous stages of the parasite. No other coccidial cysts or their stages are known to attain such dimensions. That the parasites are not sarcocysts, besnoitia or toxoplasms, require no emphasis.

It is not within the scope of this communication to consider the pros and cons of the several fine concepts and arguments advanced from time to time in respect of the zoological status of *Globidium*. The different endogenous stages of parasite described in this study resembled to a large extent those reported by Henry and Masson (1932), and Enigk (1934) in respect of *Globidium cameli*. Henry and Masson (1932) stated that no asexual forms of parasite were observed in the cases studied by them. Reichenow (1953) considered that the alleged merozoites of Enigk (1934) were actually microgametes. In the specimens examined by us, the microgametes appeared to be distinctly different in morphology from the merozoites : the former were in the form of short stumpy rods like those of *Eimeria bovis* (Hammond *et al.*, 1946) or spherical, minute granular bodies similar to the microgametes of *Globidium travassosi* (Reichenow, 1953), while the merozoites were fine and crescent shaped. Since biological tests were not carried out, these statements have their obvious limitations.

Another point which might merit attention is that the different endogenous stages were seen to develop in the same segment of small intestine side by side. This is in contradistinction to the observations of Hammond *et al.* (1946) and Reichenow (1953) who stated that the gametocytes of *Globidium* species were generally found to develop in a farther, much lower, segment of the alimentary tract while the asexual forms were confined to the anterior regions.

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