CASE OF NEONATAL CAMEL COLISEPTICEMIA IN KENYA

S. Bornstein[1] M. Younan[2] R. Feinstein[3]

Key words: Dromedary - Young animal - *Escherichia coli* - Septicemia - Kenya.

Mots-clés : Dromadaire - Jeune animal - *Escherichia coli* - Septicémie - Kenya.

■ Introduction

High mortality has been reported in neonatal camel calves (3). In East Africa a morbidity of 30% has been recorded (4) with nearly 100% mortality if there is no immediate veterinary intervention, which is often the case given the inaccessible areas where camels are kept. While neonatal diarrhea caused by *Escherichia coli* has been repeatedly described in the camel (5), there is no report on septicemic *E. coli* infections in camel calves.

■ Materials and methods

In a breeding herd of 75 female camels of the Somali type in Northern Kenya, 4 out of 10 calves aged 1-2 weeks died after showing anorexia, diarrhea and general weakness at the beginning of the 1999 calving season. A one-week old male calf was found dead on the evening preceding the authors' visit. The calf had been unthrifty for 2-3 days without developing any signs of diarrhea.

Necropsy of the calf was carried out within 14 h after death. Swabs were taken from heart blood, pericardial fluid, lungs, spleen and tonsils and stored in Stuart's medium. In addition, the prescapular and axillary lymph nodes, bone (rib), kidney, ligatured segments of jejunum and colon, respectively, as well as the ileocecal lymph nodes were taken as samples. The swabs and specimens were transported to the laboratory in a portable refrigerator and cultured on the same day.

The swabs and organ specimens were inoculated aerobically on blood agar (BA, blood agar base No. 2, Oxoid CM 271, with 5% defibrinated sheep blood). After overnight incubation at 37°C the direct cultures on BA were examined and reincubated for an additional 24 h. Anaerobic incubation was carried out for intestinal contents, kidney and ileocecal lymph node on BA, incubated in an anaerobic jar for 48 h. Isolated coliform colonies were differentiated according to standard procedures.

Serotyping of isolated *E. coli* was performed using 17 serotypes: 06, 08, 09, 020, 045, 064, 098, 0101, 0115, 0138, 0139, 0140, 0141, 0147, 0149, 0157 and 0X46 (at the Department of Bacteriology, SVA, Uppsala, Sweden).

Pieces of brain, heart muscle, kidneys, lungs, liver, spleen and tonsils were fixed in 10% buffered formaline. After routine histological processing, sections were cut and stained with hematoxylin and eosin.

■ Results

At necropsy, fibrinous fluid was found in the pericardium. The epicardium, endocardium and renal pelvis displayed petechiae. All body lymph nodes and tonsils were hyperemic and enlarged. The liver was very pale and hard. The rectum contained pasty whitish feces and the mucosa of the intestines, particularly the colon, was hyperemic and thickened. The meninges were slightly hyperemic.

The aerobic bacteriological investigation revealed the presence of *E. coli*, isolated in pure cultures from the body lymph nodes, tonsils, spleen, lungs, bone marrow, heart blood and pericardial fluid. In addition, pure growth of *E. coli* was obtained in anaerobic culture of the ileocecal lymph node. The anaerobic incubation of kidney and intestinal contents produced a clear predominance of growth of *E. coli* intermixed with few solitary (less than seven) clostridial colonies showing the typical morphology of *Clostridium perfringens*. Isolated *E. coli* did not agglutinate any of the tested sera.

At the histological level, the main findings were intestinal changes characterized by marked dilatation of goblet cells and moderate to severe mucosal infiltration of leukocytes, showing a predominance of eosinophils in the small intestine (figure 1). The large intestine exhibited a severe hyperemia and markedly dilated crypts with excessive amounts of mucus. In restricted areas the surface epithelium was eroded (figure 2). The intestinal changes were diagnosed as acute catarrhal enteritis. Parasites were not found.

Body lymph nodes (figure 3), spleen (figure 4) and tonsils displayed a severe lymphocytic depletion. In these organs, lymphatic follicles and germinal centers (B-cell areas) were not observed. The heart presented degenerative changes in the fibers of the conducting system. The main features observed were pronounced intracellular edema and precipitation of chromatin in thick, coarse granules or pyknosis. The other tissues histologically examined presented circulatory changes: hyperemia, congestion and small hemorrhages. No inflammatory infiltrates were seen in these latter organs.

1. Department of Parasitology (SWEPAR), National Veterinary Institute (SVA), Uppsala, Sweden

2. University of Egerton, Njoro, Kenya

3. Department of Pathology, SVA, Uppsala, Sweden

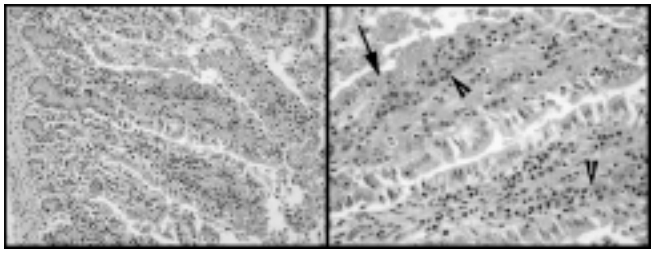

Figure 1: *Necrosis and inflammation of the small intestine (left); Detail of small intestinal mucosa showing infiltrated eosinophils (arrow heads). Sharp demarcation between seemingly intact surface epthelium and necrotic area (arrow) (right).*

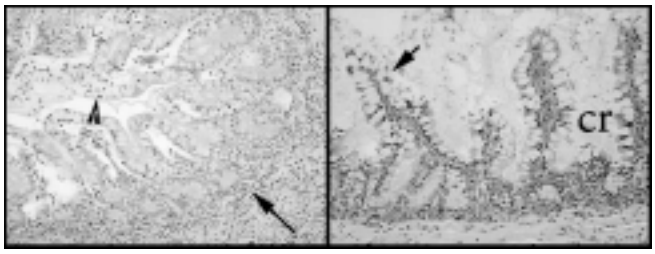

Figure 2: *Large intestine showing dilated epithelial cells, desquamation of the epithelial lining (arrow head) and infiltrated leukocytes most prevalent in the basal mucosal areas (arrow) (left); Large intestine displaying vacuolar degeneration of the epithelium and severely dilated crypts (cr) containing excessive amounts of mucus. Slight leukocytic infiltration in the basal mucosal areas (right).*

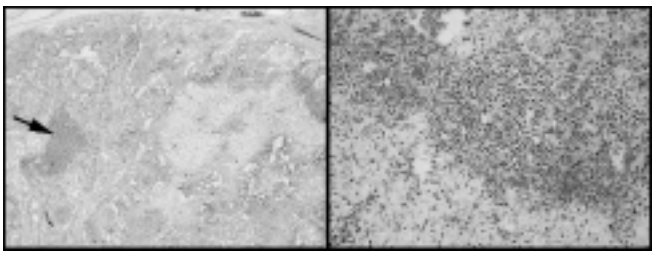

Figure 3: *Lymph nodes showing pronounced reduction of lymphoid follicles (arrow) (left); Lymph nodes showing markedly depleted lymphoid follicles and hemorrhages (right).*

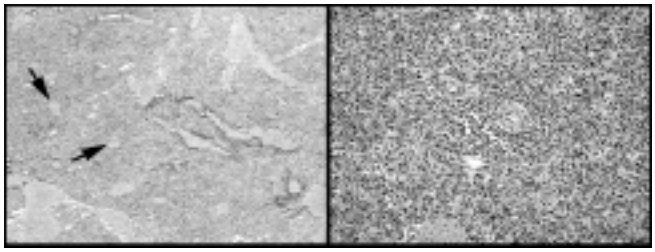

Figure 4: *Spleen exhibiting markedly reduced white pulp due to a severe lymphatic depletion. Small-sized periarteriolar lymphoid sheaths (PALS, arrows) (left); Spleen, closer view of depleted PALS (right).*

■ Discussion

Clinical history and postmortem findings as well as age of the affected calf differ from the common findings in *E. coli* diarrhea of camel calves (5). Bacteriological and pathological findings indicate that the calf had a septicemic condition (*E. coli* infection) resembling the commonly observed condition in neonatal bovine calves during their first week of life (1). The severe lymphocytic depletion seen in lymphatic organs indicates some kind of strain on the immune system of the calf.

Traditional animal husbandry practices of some camel keeping people often result in inadequate intake of colostrum by the newborn calf (1, 2). Failure to receive sufficient colostrum early in life may result in agammaglobulinemia or hypogammaglobulinemia, conditions which are considered prerequisite to the disease (2).

REFERENCES

1. GYLES C.L., 1993. *Escherichia coli*. In: Gyles C.L., Thoen C.O., Eds., Pathogenesis of bacterial infections in animals, 2nd ed. Ames, IA, USA, Iowa State University Press.

2. HULSEBUSCH C., 1999. Immunoglobulin G status of camels during 6 months postnatum. Doct. Thesis, Stuttgart, Germany, Center for Agriculture in the Tropics and Subtropics, University of Hohenheim.

3. KHANNA N.D., TANDON S.N., SAHANI M.S., 1992. Calf mortality in Indian camels. In: Allen W.R., Higgins A.J., Mayhew I.G., Snow D.H., Wade J.F., Eds., Proc. 1st Int. Camel Conference, Dubai, United Arab Emirates, 2-6 February 1992. Newmarket, UK, R&W Publications, p. 89-92.

4. SCHWARTZ H.J., DIOLLI M., Eds., 1992. The one-humped camel in Eastern Africa. A pictorial guide to diseases, health care and management. Berlin, Germany, Verlag Josef Margraf.

5. WERNERY U., KAADEN O.-R., 1995. Infectious diseases of camelids. Berlin, Germany, Blackwell Wissenschafts-Verlag.