

Histopathological Studies of *Eimeria bovis* Infection in Calves

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Abstract

Six experimentally infected calves were slaughtered at 15 days post infection (dpi) (Group A), 20 dpi (Group B), 25 dpi (Group C) and the intestinal mucosa was examined for pathological changes. The affected part of the intestine was removed, opened and examined for macroscopic findings. The animals studied showed lesions of varying severity. The gross pathology revealed no remarkable abnormal findings in calves of group A. Redness of the mucosa of the caecum and colon in six calves of group B and C was noted. There were no first generation schizonts evident grossly. The main characteristic under the light of microscope of the intestinal sections taken from the calves examined during the late prepatent period were mild inflammatory infiltration of the mucosa and increased cellular debris, parasitic structures were identified from small and large intestine. The caecum and colon showed particularly high levels of infection.

Keywords: *Eimeria bovis*; Calves; Histopathology

Introduction

Eimeria bovis and *E. zuernii* are the economically most important of the twenty-one or so coccidial species infecting cattle [1]. At least nine species of coccidia have been reported in cattle which include *Eimeria subspherica*, *E. zuernii*, *E. alabamensis*, *E. elipsoidalis*, *E. cylindrica*, *E. bovis*, *E. condensens*, *E. bukidonensis* and *E. auburnensis* [2]. Two species i.e. *Eimeria zuernii* and *Eimeria bovis* are more pathogenic and common [3]. All *Eimeria* spp. share a similar monoxenous life cycle with an internal (parasitic) and external (environmental) phase. They are strictly host specific and develop within cells at certain sites of the intestinal mucosa. Most studies into the biology of cattle coccidia have been carried out with *E. bovis*. Under *in vitro* conditions sporozoites liberated from *E. bovis* oocysts may invade various cell types of different hosts and certain (unknown) target molecules allowing recognition, adhesion and subsequent penetration have been postulated. However, further development was exclusively observed in bovine cells and few gamonts and oocysts were obtained only in fetal gastrointestinal cells [4]. In the natural host sporozoites traverse mucosal cells without considerable alterations and finally invade endothelial cells of the central lymph capillaries of the ileal villi [5]. The amount of excreted oocysts increases with the infective dose until a certain level above which inoculation of more oocysts does not further increase but may even decrease oocyst production, an effect termed crowding phenomenon [6]. Depending on moisture, temperature and other environmental factors the oocysts sporulate and may reach infectivity within a week. The sporulated oocysts contain four sporocysts each harbouring two sporozoites. Under favourable conditions (moisture, temperature of 5°C to 8°C) they maintain infectivity for several months and may even survive the winter season [7]. Keeping in view the importance of disease in calves, this research project was designed to describe the lesions associated with experimental *E. bovis* infection in cattle and attempts to elucidate the pathogenesis of this protozoal disease.

Materials and Methods

Histopathological studies were conducted at Department of Pathology, of University of Veterinary and Animal Sciences, Lahore.

Six experimentally infected calves were slaughtered at 15 days dpi

(Group A), 20 Days dpi (Group B), 25 days dpi (Group C) and the intestinal mucosa was examined for pathological changes. The affected part of the intestine was removed, opened and examined for macroscopic findings. Samples were collected from different localizations (jejunum, ileum, caecum, and colon) and fixed in 10% buffered formaldehyde solution. Specimens were trimmed, embedded in paraffin wax, and cut at a thickness of approximately five (5) micrometers. All slides were stained with haematoxyline and eosin stain.

Staining of tissues [8]

- Deparaffinized sections, hydrated through graded alcohols to water.
- Stained with Ehrlich's haematoxylin for 30 min.
- Washed well in running tap water.
- Differentiated with 1% acid alcohol (1% HCl in 70% alcohol), for 5-10 seconds.
- Washed blue well in running tap water until sections are again blue (10-15 minutes).
- Stained with 1% aqueous Eosin, for 10 min.
- Washed in running tap water for 1-5 min.
- Dehydrated, cleared, and mounted in D.P.X

Parasitic stage was identified through histopathological studies of the intestinal mucosa by light microscopy [9].

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Results

The animals studied showed lesions of varying severity. The gross pathology revealed no remarkable abnormal findings in calves of group A. Redness of the mucosa of the caecum and colon in six calves of group B and C was noted. There were no first generation schizonts evident grossly. The main characteristic under the light of microscope of the intestinal sections taken from the calves examined during the late prevalent period were mild inflammatory infiltration of the mucosa and increased cellular debris, parasitic structures were identified from small and large intestine. The caecum and colon showed particularly high levels of infection. In calves of both groups inflammatory granulocytic infiltration of the mucosa and cellular debris in most of the intestinal portion were seen.

In group C, calves slaughtered at 25 dpi, the intestinal mucosa was characterized by shortened villi or necrosis and degeneration of villi. Haemorrhages in mucosa and sub-mucosa were also observed. Some of the glands in the sub-mucosa of intestine showed degeneration and necrosis.

Tips of few villi showed necrosis, haemorrhages in the mucosa of intestine, minor infiltration of leukocytes in the muscles. Villi and crypts of lieburkuhn are widely separated from each other and mild leucocytic infiltration and edema are seen in the lamina propria.

In both animals regeneration and or hyperplasia of the cryptal epithelium with proliferation of goblet cells was observed in large intestine. At most of localizations examined, mixed cellular inflammatory infiltrates in the mucosa and cellular debris were still present (Figures 1 and 2).

Discussion

In histopathological studies showed an inflammatory granulocytic infiltration of the mucosa and cellular debris in most of intestinal portion. The most changes were observed in intestines of the calves sacrificed during patent period of the infection. In those calves acute typhilitis and necrotizing colitis and parasitic infection, particularly high in the calves. Similar findings were reported by Mundt et al. [9] who conducted studies in *E. zuernii* infection model in order to investigate the pathology of *E. zuernii* coccidiosis. Six of these calves underwent pathological examination at various points. Significant macroscopic and microscopic changes were demonstrated. Parasitic stages were identified in the intestinal mucosa of infected calves during the late prepatent and patent periods. Similar findings were also

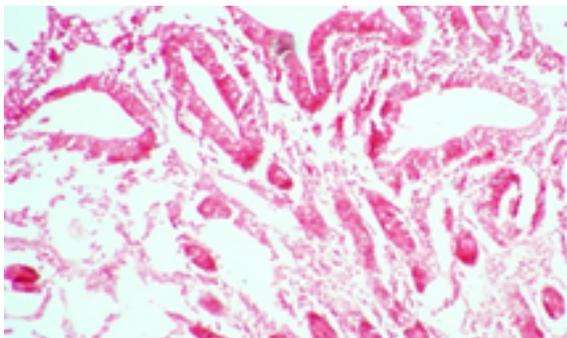


Figure 1: Small intestine from a calf. Villi and crypts of lieburkuhn are widely separated from each other and mild leucocytic infiltration and edema are seen in the lamina propria (X-100).

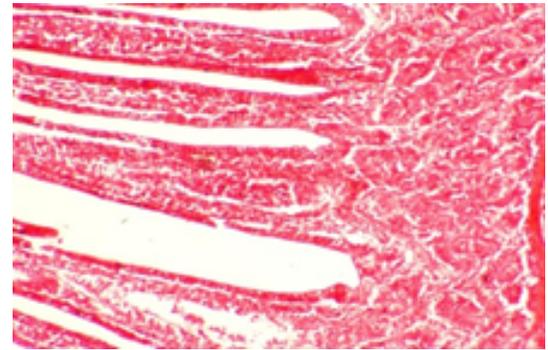


Figure 2: Small intestine of calf. The lamina propria is hyper cellular due to infiltration of inflammatory cells.

reported by Stockdale [10], who infected calves with *E. zuernii* oocysts and demonstrated the presence of Schizonts in the lamina propria of the small intestine with only little inflammatory reaction, and lesions in caecum and colon were similar to each other and most extensive between 18 and 21 days with loss of the epithelium and a presence of a thick layer of epithelium and a presence of thick layer of Fibrin in which cellular debris and bacterial colonies were incorporated. After 21 dpi, the mucosal regeneration started. Similar findings were also reported by Chandler and Read [11]. They reported that the severe attacks by pathogenic species of genus *Eimeria* in the gut of affected animals caused great destruction of the epithelium along with severe haemorrhages and sloughing off the walls.

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