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Research Article

PATHOLOGICAL STUDIES ON DIFFERENT ISOLATES OF EIMERIA TENELLA INFECTION IN CHICKENS

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ABSTRACT

Chickens infected with three different isolates of *Eimeria tenella* revealed that the severity of the clinical signs and amount of blood in faeces were maximum due to isolate-III followed by the isolate -II. Pathological studies revealed that coccidial infection caused changes of haemorrhagic typhlitis and atrophy of bursa of Fabricius. It is concluded that the isolate-III was the most pathogenic followed by the isolate -II and isolate-I.

Key words: *Eimeria tenella*, chickens, clinicopathology, typhlitis

Caecal coccidiosis is caused by *Eimeria tenella* and results in heavy economic losses due to high morbidity and mortality (McDougald and Reid, I991). It has been reported that birds infected with the same number of oocysts of different isolates of *E. tenella* showed different degrees of pathogenicity of coccidiosis as evidenced by differences in mortality and clinical signs (Fitz-Coy, I992). In India, various isolates of *E. tenella* reported from outbreaks of caecal coccidiosis in different poultry farms have shown different degree of drug resistance (Yadav and Gupta, 2001). Due to paucity of literature, the present study was undertaken to investigate sequential pathological changes in broiler chicks infected with different isolates of *E. tenella.*

MATERIALS AND METHODS

A day-old sixty broiler chicks of Ross strain were obtained from a local hatchery and reared in well ventilated rooms under hygienic conditions. All birds were given ad libitum coccidiostat free standard chick feed and provided clean drinking water throughout the experiment. At the age of two weeks the chicks were randomly divided into four groups of 15 birds each. The birds of groups I, II and III were inoculated with sporulated oocysts of I, II and III isolates of *Eimeria tenella* collected from three different outbreaks, respectively @ $1\times I0^5$ sporulated oocysts/bird (McDougald and Reid, I991) in 1 ml NSS by intra crop intubation method whereas the birds of group IV which served as controls were given only 1ml NSS. Each bird was closely observed daily for clinical signs and mortality, if any.

Three birds from each subgroup were sacrificed on the day of start of the experiment and subsequently on days 3, 5, 7 and 14 post infection (PI) and were thoroughly examined for gross lesions, if any. A portion of caecum, small intestine, liver, lungs, kidneys, bursa of Fabricius, spleen and thymus, was collected in 10% neutral buffered formalin. The fixed tissues were processed for paraffin embedding and sections of 3-4 µ were stained with haematoxylin and eosin (Luna, 1968).

RESULTS AND DISCUSSION

Clinical signs of caecal coccidiosis observed in all the infected groups were anorexia, depression, dullness, growth retardation, drooping of wings and ruffled feathers from day 4 PI and blood in faeces from day 6 which became at peak on day 7 PI. The severity of clinical signs

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and the amount of blood in faeces were the maximum in group III followed by groups II and I. Two birds died only in group III on day 7 PI indicating that severity of the infection was of higher magnitude in isolate III. The blood in faeces started to decline from day 8 PI and reached almost to negligible on day I0 PI. The above clinical signs were almost similar to those reported by other workers (Witlock, 1983, McDougald and Reid ,1991, Panda *et al.*, 1997) in chickens suffering with caecal coccidiosis.

Gross pathological changes following infection with different isolates of *E. tenella* in chickens were presence of catarrhal exudate in caeca on day 3 PI. On day 5 PI in all the infected groups, caeca appeared swollen due to presence of varying amount of blood mixed exudate in lumen along with haemorrhagic spots on the caecal mucosa. The lesions became more severe on day 7 PI with extensive haemorrhagic and necrotic spots visible from serosal surface of caeca (Fig 1) particularly in group III and presence of clotted and unclotted blood exudate in the lumen. The overall severity of the caecal lesions was maximum in group III followed by group II and I. On day 14 PI, mucosal surface of caeca was smooth in groups I and II whereas it was rough and raised in group III.

On histopathological examination, caecal mucosa of all the birds in infected groups on day 3 PI, showed hyperplasia of lining epithelium and infiltration of heterophils and mononuclear cells in lamina propria. A large number of first stage developing schizonts containing immature merozoites were noticed within the vicinity of mucosal epithelium. The groups of schizonts were also seen in lamina propria. These lesions were most severe in birds of group III and moderate with almost same magnitude in groups I and II. In addition, there were thining of epithelial lining of mucosa and mild haemorrhages at places in the birds of group III. On day 5 PI, caeca of all the infected groups revealed different degrees of haemorrhagic typhlitis characterized by extensive inflammatory cells infiltration consisting of heterophils, mononuclear cells and eosinophils, degeneration of crypts and lining epithelium and haemorrhages in the mucosa along with oedema and infiltration of inflammatory cells in the submucosa. Mucosal haemorrhages were maximum in the birds of group III and least in group I. Additionally, severe haemorrhages were also noticed in submucosa of chickens in group III. Clusters of second stage schizonts which contained a large number of mature and immature merozoites (Fig 2) were noticed in caecal mucosa of all infected groups.

On day 7 PI, microscopic changes differed extensively in different isolates. In group III, there was complete loss of architecture of caecal mucosa along with the presence of haemorrhagic exudate into the lumen. The contents of the exudate were inflammatory cells, erythrocytes, cellular debris and oocysts. The mucosa revealed severe haemorrhages, inflammatory cells infiltration consisting of macrophages, lymphocytes and heterophils, destruction of most of the glands and presence of large number of oocysts along with sloughing of mucosal epithelium. Haemorrhages and inflammatory reaction were also extended to submucosa (Fig 3) and muscular layer. In a few cases, muscularis mucosa was completely damaged and there was no line of demarcation between mucosa and submucosa. The lesions in groups I and II were less severe. The mucosal lining was intact, at places, and in mucosa, erythrocytes was comparatively less and muscular layer was intact. On day 14 PI, caeca of group III showed presence of granulation tissue along with macrophages and degenerated oocysts (Fig 4). A few regenerated crypts in the mucosa were also observed. In groups I and II, caeca revealed almost complete regeneration of lining epithelium and presence of number of regenerated crypts and mononuclear cells in the mucosa.

Gross and histopathological changes following infection with different isolates of *E. tenella* in chickens on day 3 PI corresponded to the development of first generation of schizonts and were almost similar to those reported by McDougald and Reid, 1991. On day 5 PI, pathological findings clearly revealed that maturation of second generation schizonts is accompanied by excessive tissue damage, bleeding and disruption of the caecal glands. The

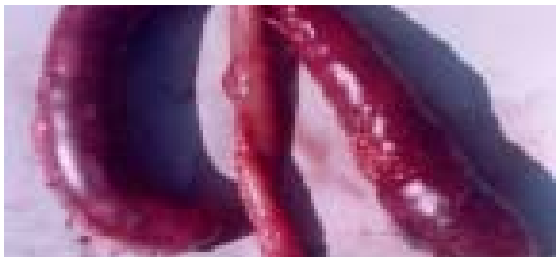

Fig 1. Caeca of isolate III infected chick showing swelling and presence of haemorrhagic and necrotic spots visible from serosal surface on day 7 PI.

Fig 2. Caecum of isolate-III infected chick showing typhlitis along with a number of schizonts containing mature and immature meroziotes in mucosa on day 5 PI. (H. & E. x 200)

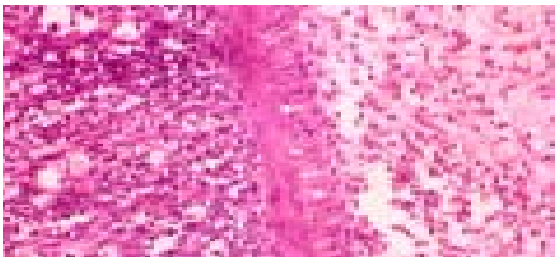

Fig 3. Caecum of isolate-III infected chick showing leucocytic infiltration and oocysts in mucosa along with haemorrhages, heterophils and oedema in submucosa on 7 day PI (H. & E. x 200)

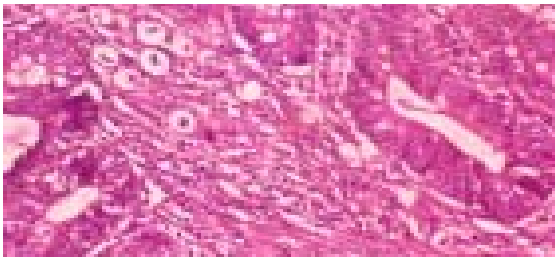

Fig 4. Caecum of isolate-III infected chick showing granulation tissue alongwith a few regenerated crypts and degenerated oocysts on day 14 PI (H. & E. x 200)

overall severity of the caecal lesions on days 5 and 7 PI was maximum in group III followed by group II and I. Pathological changes in caeca due to coccidiosis could be explained to some extent on the basis of interaction of the parasites with host immune system (Lillehoj and Lillehoj, 2000). Allen (1997) reported a significant increase in nitric oxide production in chickens infected with *E. tenella* and correlated to development of caecal lesions.

Liver sections in group III exhibited small focal areas of necrosis, infiltration of heterophils and mononuclear cells on days 5 and 7 PI. On day 14 PI, liver did not reveal any significant change in all the infected groups.

All the infected groups revealed mild depletion of lymphocytes and presence of few heterophils in follicles of bursa of Fabricius on day 5 PI. On day 7 PI, group III also exhibited atrophy of a few follicles and increase in inter follicular space in the bursa due to reticular cells proliferation and mild mononuclear cells infiltration, reflecting immunosupessive effect of the disease. These results are in accordance with the findings of Paulos *et al*., (1997). On day 14, the bursal follicles did not exhibit any lesion in all the infected groups.

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