Clinical, paraclinical and morphopathological aspects in cecal eimeriosis of broilers

Aspecte clinice, paraclinice și morfopatologice în eimerioza cecală a puilor de găină

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ABSTRACT

Investigations were conducted at AVICOLA Trade Company of IAȘI, during March 2008-January 2009, on five broiler series belonging to Ross 308 and Cobb 500 breeds, bred on permanent layer, for pointing out the appearance and the clinical evolution of eimeriosis episodes, identification of incriminated species and morpho-pathological changes generated by the parasitic aggression. Faeces, sampled from chicken cloaca and on the floor, were analysed by the qualitative flotation method (Willis) for identifying *Eimeria* species. The necropsic examination has determined the intestinal segment affected by eimeriosis. Clinically, cecal eimeriosis has started as acute stage in 10 until 42 day old chickens, as episodes dominated by enteroragy incidences, bloody diarrhoea, anaemia, neurologic troubles and, finally, death. Paraclinically, we have identified some species of *Eimeria* genus that parasitized the small intestine, dominated by *Eimeria tenella*, found in the caecum. Morpho-pathologically, we found a growth of the caecum with turgescent aspect and reddish-violaceous coloured wall. Caecum opening allowed the release of great amounts of uncoagulated or coagulated blood, while severely affected-cecal mucous had bloody and bloody-necrotic typhlitis.

**Keywords:** broiler, caecum, *Eimeria tenella*, bloody typhlitis

Introduction

Eimeriosis of poultry broilers is a Sporozoa produced by numerous species belonging to *Eimeria* genus, which is located in the segments of small and large intestines, clinically expressed by enteric syndrome, enteroragy, neurological troubles, body weakening and, finally, death. Species from *Eimeria* genus act simultaneously, affecting large areas of the digestive tube (duodenum, (*E. acervulina, E. mitis, E. mivati*), jejun (*E. maxima*), ileon and rectum (*E. necatrix, E. hagani, E. praecox, E. brunetti*), caecum, (*E. tenella*), cloaca (*E. brunetti*)), having destructive and convergent effect. The effects are destroying when the toxigene species from the small intestine is associated with *Eimeria tenella* from caecums. The simultaneous evolution of intestinal and cecal eimeriosis results in starting and the evolution of some acute clinical episodes with high mortality (1, 2, 7). The aggression of *Eimeria tenella* species is severe and rapid because of schizogonic processes developed in the cecal epithelium they destroy by generating a severe cecal haemorrhage. The appearance of clinical episodes is conditioned by the interaction between the factors depending on the host body, parasite and microclimate, provided by growing technology.

Breeding broilers in halls on permanent layer favours sporogenesis of immature oocysts, which are eliminated by digestive way by sick or carrier broilers that eliminate parasites, preserving the mature oocysts on the floor, for a longer period, for successive chicken series. The variation of microclimate factors influences the duration of the biological cycle of *Eimeria*, modifying the duration of parasite development stages and the atypical outburst of some clinical episodes (2).

The goal of these investigations was to detect the starting moment and the evolution of eimeriosis episodes in broilers bred on permanent layer in successive series, identification of
incriminated species, clinical expression and dimension of morpho-pathological changes.

**Materials and methods**

The investigations were conducted during March 2008 - January 2009 on one broiler series belonging to Ross 308 and Cobb 500 breeds, bred on permanent layer at AVICOLA Trade Company of IAŞI. The chickens were examined clinical, paraclinical and morphopathological. The faecal samples were collected from the chickens cloaca and from the floor of the halls where it have been recorded clinical cases. The coproscopic examinations were performed in the Laboratory of Parasitology of the Faculty of Veterinary Medicine Iasi. The necropsy examination was performed daily in the necropsy room of the unit taken in the study. The microscopic images were microphotographed and macroscopic aspects were taken with a digital camera.

**Results and discutions**

In the group considered for the study, the eimeriosis evolved acute and subacute, affecting episodically chicks from the age of 10 days to 42 days. These aspects show that chickens during the growth period are likely to be infected with *Eimeria* at any age. The infection is facilitated by microclimate conditions favorable for the exogenous development of species of the genus *Eimeria*. After a variable incubation period of 5-10 days, the chicks were observed with acute symptoms, characterized by polydipsia, loss of appetite, adynamy, horiplumation, mucosa palidity, continuous peep, anorexia, diarrhea, bleeding (figs. 1, 2) support on the wings during movement, ataxia, limb paralysis and death (figs. 3, 4, 5, 6).

**Fig. 1, 2.** Cecal eimeriosis in 18 days old chicken with bloody diarrhoea, in the hall, on permanent layer

**Fig. 3.** Cecal eimeriosis in 32 day old chicken found in condition of prostration, feather rising, lack of dynamism, anaemia

**Fig. 4.** Cecal eimeriosis in 32 day old chicken with neurotical symptoms and diarrhoea with bloody faeces
The cecal eimeriosis evolves acute at the chickens of all ages, but it is expressed clinically by adinamy, anorexia, anaemia, paralysis of limbs, hemorrhagic diarrhea and fatal outcome at most of the chicks from 18 to 32 days (figs. 5, 6).

The hall was also observed hemorrhagic feces on the bedding and chickens with hemorrhagic diarrhea, without being present other symptoms (figs. 7, 8).

The infestation of the chickens was performed by ingestion of a mixture of species in which the species *Eimeria tenella* was dominant, imposing its pathogenicity.

The coproscopic qualitative examinations revealed oocysts of variable shapes and sizes which belonged to other species of the *Eimeria* genus, located in the small intestine and caecum (figs. 9, 10).

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The necropsic examination of the chickens infected with *Eimeria tenella* showed the anemia syndrome caused by abundant bleeding in caecum.

The examination of the abdominal cavity revealed the shape and volume changes of the caecum, the most affected segments of the digestive tract (figs. 11 to 18).

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**Fig. 9.** *Eimeria* spp.: immature oocysts sampled from sick chicken cloaca (Willis Method, 10 x 10)

**Fig. 10.** *Eimeria* spp.: immature oocysts sampled from faeces spread on layer (Willis Method, 10 x 10)

**Fig. 11.** Cecal eimeriosis in 10 day old chicken: bloody typhlitis

**Fig. 12.** Cecal eimeriosis in 12 day old chicken: bloody typhlitis

**Fig. 13.** Cecal eimeriosis in 14 day old chicken: bloody typhlitis

**Fig. 14.** Cecal eimeriosis in 16 day old chicken: bloody typhlitis
The opening of the caecum at 4 days after infection revealed extensive bleedings in caecum, while in the 6th and the 7th day, the bleeding contents occurred as clots. The cecal wall is edematiated, infiltrated and the mucosa presented hemorrhagic typhlitis with extensive destruction of the epithelium, exudation and penetrations of the basement membrane (Fig. 19, 20, 24).
Fig. 19. Cecal eimeriosis in 18 day old chicken with bloody typhlitis; uncoagulated blood.

Fig. 20. Cecal eimeriosis in 20 day old chicken with bloody typhlitis; coagulated blood.

The caecum wall appeared thickened edematized, infiltrated, acquiring a reddish color with white spots or yellow-white in May and hard to palpation after 7-8 days after infection. The opening of cecal wall revealed hemoragico-necrotic typhlitis with hyperplasia of the chorionic elements and exudation of variable intensity, bleeding areas with different strains (5) (figs. 21, 22, 23).

Fig. 21. Cecal eimeriosis in 27 day old chicken: bloody typhlitis, bosselated cecal loops, tough on palpation.

Fig. 22. Cecal eimeriosis in 27 day old chicken bloody-necrotic typhlitis; oedema of intestinal glands, focal necroses.
Fig. 23. Cecal eimeriosis in 34 day old chicken: intestinal and cecal.

Fig. 24. Cecal eimeriosis in 34 day old chicken: bloody typhlitis: content as coagulum, thicker edematized wall, completely destroyed mucous.

The examination of the mucosa scrape showed different stages of schizogenic and gametogenic development: trophozoits, schizonts with merozoits and immature oocysts which confirmed eimerian etiology of clinical episodes. The presentation of the lesions according to the age of chicks suggests the extending of cecal eimeriosis throughout the whole breading period of the chickens and the nature of tehnopaty of eimeriosis, aspects which require a particular attention in preventing the outbreak of clinical episodes which often leads to great economic losses.

_Eimeria tenella_ colonize the cecal mucosa causing the swelling of the walls and the appearance of small reddish outbreaks, visible through the traversal of serosa. The increasing of the pathogen process causes conflation of the bleedings and imprints red-purple or dark red color to the cecal bags. The opening of the caecum reveals hemorrhagic infiltration mucosa and the accumulation of exudate into the lumen in the form of structured clots. The massive hemorrhage of caecum are the main cause of anaemia and death of chickens. In moderate infections, in the caecum it is observed dehydrated, brittle clots with fibrin remains and adherents more or less at the hyperplasia of the mucosa (1, 2, 5). The mucosal hyperplasia is induced by intense cellular reactions that take place in the mucosa and submucousa (3).

The induced immune reactions are characterized by rapid change in the proportion of intraepithelial T lymphocyte subpopulations, thus it is point out a increase of CD4+ lymphocyte by 20% on the eighth day after infection and of CD8+ lymphocyte by 10%. The T lymphocyte and macrophages are involved in the transepithelial migration of parasites and their location in lamina propria (6, 8). Both types of cells found in the side "drawers" of M cells, cells specialized in antigen capture and in their presentation to the cells that produce the immune response (5).

The severe infections with _Eimeria tenella_, with cytological changes and tissue damages, disturb the intestinal biocoenosis encouraging the risk of infections with _Salmonella, Clostridium, Escherichia_, generating large economic losses (6). Cecal eimeriosis of chickens produced by _Eimeria tenella_ is a morbid complex resulted from the interaction of parasites, bacterial microflora, the reactivity of the host and microclimate factors.

**Conclusions**

The eimeriosis of broiler series belonging to Ross 308 and Cobb 500 breeds, bred on permanent layer, in five successive series was dominated by Cecal eimeriosis produced by the _Eimeria tenella_.

The clinical episodes evolves acutely from the age of 10 days to 42 days, characterized by enteroragie, hemorrhagic diarrhea, anaemia, state of prostration, anorexia, adynamy, limb paralysis, followed by death.

Laboratory it have been identified also other species of the genus _Eimeria_ located in the intestines, but _Eimeria tenella_ species prevailed in both in the samples taken from poultry and from the permanent layer.

The necropsy examination revealed increased volume of the caecum, with red-purple coulored wall, filled with hemorrhagic content in all examined cases, regardless of age and the lesions of hemorrhagic or hemorrhagic-necrotic
typhlitis of different intensities. Histological it were outlined the schizogonic stages (trofozoits, schizonts) and the immature oocysts which confirmed the eimeric etiology of bleeding CECA.

The aggression of the species Eimeria tenella was severe and rapid, causing death in chickens of all ages and economic loss record.

REZUMAT


Cuvinte cheie: pui broiler, cecumuri, Eimeria tenella, tiflită hemoragică

References