



Evaluation of coccidiosis vaccines in chicken

Haythm Sharaban ^{a*}, Mohamed Seadawy ^b, Fares El-khayat ^a, Abd El-Galil El-Gohary ^a

^a Department of Poultry and Rabbit Diseases, Faculty of Veterinary Medicine, Kafrelsheikh University, Egypt.

^b Main Chemical Laboratories Egypt Army, Egypt.

*Correspondence: haythamsharaban@yahoo.com

Abstract

Coccidiosis is a serious disease affecting poultry. It is caused by a protozoan parasite of genus *Eimeria* that occupies the intestinal tract, causes tissue damage, and results in interruption of feeding and high mortality. This study aimed to evaluate the effect of different types of coccidial vaccines on the prevention of coccidiosis in chicken under field conditions. Chicks (n = 12; one-day-old; Avian-48 broiler) were randomly divided into 6 groups (G1, G2, G3, G4, G5, and G6), each group contained 20 chicks. The negative (G3) and positive (G6) control groups were non-vaccinated, while the remaining groups (G1, G2, G4, and G5) were vaccinated by live attenuated vaccine A (precocious strains, G1 and G4) and live non-attenuated vaccine B (wild strains, G2 and G5). G4-G6 were challenged on the 28th day by 1×10^5 sporulated oocysts of *Eimeria tenella*. The feed conversion rate (FCR), body weight gain (BWG), oocyst shedding, lesion score, oocyst index, and histopathology were observed and recorded in all groups. Vaccinated challenged groups (G4 and G5) had significantly lower FCR, oocyst count, and oocyst index but with higher BWG than the non-vaccinated challenged group (G6). Interestingly, G4 and G5 had lower lesion scores with no mortality as compared to G6 which showed 10% mortality. This study concludes that the usage of the anti-coccidial vaccine has significant protective efficacies in broilers with great potential with attenuated strain vaccine.

Keywords: Coccidiosis; Broiler; Oocyst shedding; Oocyst index; Lesion score; Vaccination.

1. Introduction

Coccidiosis is one of the most important and dangerous diseases affecting poultry production. The protozoan of the genus *Eimeria* occupies the intestinal tract and causes tissue damage, which results in interruption of feeding, digestion, and nutrient absorption; dehydration; blood loss; loss of skin pigmentation, and increased susceptibility to other diseases. The clinical symptoms vary between growth retardation, watery feces, necrotic enteritis, hemorrhagic enteritis (Jaipurkar et al., 2002). The disease may be mild, resulting from the ingestion of a few oocysts and may escape unnoticed, or it may be severe as a result of the ingestion of millions of oocysts. Most infections are relatively mild, but because of the potential for the disastrous outbreak and the resulting financial loss, almost all young poultry are given continuous medication with low levels of anti-coccidial drugs, which prevent the infection or reduce to a low immunizing level (Hafez, 2008).

Drug resistance is a complex global public health challenge and no single or simple strategy will suffice to fully contain the emergence and spread of infectious organisms that become resistant to the available antimicrobial drugs. The development of drug resistance is a natural phenomenon in microorganisms and is accelerated by the selective pressure exerted by the use and misuse of antimicrobial agents in humans and animals. The current lack of new antimicrobials on the horizon to replace those that become ineffective brings added urgency to the need to protect the efficacy of existing drugs (WHO, 2014). *Eimeria spp.* resistance against

polyether ionophores (which constitute 80% of anti-coccidial use worldwide) develops slowly and may take few years (Anish Yadav and Gupta, 2001). The worldwide intensive use of anticoccidial drugs to prevent coccidiosis has inevitably led to the development of resistance to all anticoccidial drugs as long-term exposure to any drug will result in loss of sensitivity. The widespread occurrence of resistance has been described in the United States of America, South America, Europe, and China (Peek and Landman 2003; Peek and Landman 2004). Despite the widespread occurrence of resistance, at least in Europe, coccidiosis outbreaks seem to have had a limited impact so far. This is explained by the fact that resistance in many cases has allowed the occurrence of trickle infections, which are essential in the building up of immunity (McDougald and Shirley, 2009). Resistance to sulfaquinoxaline, nitrofurazone plus furazolidone, amprolium, clopidol, nicarbazin, sodium sulphadimethyl pyrimidine, and maduramicin in various field isolates of *Eimeria spp.* has been reported from north India (Agarwal et al., 2013).

Vaccines against coccidiosis have in the past been used mostly in breeder pullets and turkeys. There are three types of coccidial vaccines, the first type is subunit vaccines (composed of a purified antigenic determinant that is separated from the virulent organism). Such vaccines recombinant proteins are expressed from DNA of various developmental stages (sporozoites, merozoites, and gametes) of the *Eimeria*. No commercial products, except CoxAbic®, have been marketed to date (Dalloul and Lillehoj, 2006). The second type is non-

attenuated vaccines consist of *Eimeria* parasites, which have not been modified in any way to change their pathogenicity and originate from laboratory or field strains. Examples of such vaccines are: Coccivac®, Immucox®, Inovocox™, and Advent™ (Chapman et al., 2002). The third type of coccidial vaccines is attenuated vaccines, attenuated vaccines consist of *Eimeria spp.* strains, which have been manipulated in the laboratory in order to decrease their virulence. Reduced virulence has been performed by serial passages of the parasite in chicken embryos. Examples of such vaccines are Livacox® and Paracox® (Shirley and Bedrnik 1997). Live anti-coccidial vaccines have proved to be an effective alternative to anti-coccidial drugs for the control of chicken coccidiosis. Some live anti-coccidial vaccines, such as Coccivac®, Immucox®, Paracox®, and Livacox® have been available in the world market for several years, and these vaccines have contributed significantly to the control of chicken coccidiosis (Williams, 2002).

There is a lack of information available in the scientific research on the assessment of different coccidial vaccines used in Egypt. Therefore, this study aimed to evaluate the effect of different types of coccidial vaccines on the prevention of coccidiosis in birds under field conditions.

2. Materials and methods

The experiment was carried out in Animal Health Research Institute, Tanta branch, Egypt. All procedures were carried out in accordance with national laws and regulations for the handling of animals to avoid harm and minimize their pain.

2.1. Anticoccidial vaccine

Live attenuated vaccine (precocious strain) (A) and live non-attenuated vaccine (wild strain) (B) were used in this study. Every 1 ml of the live attenuated vaccine contains 30000-50000 oocysts of *E. acervulina*, *E. tenella*, *E. maxima*, and 10000 oocysts of *E. necatrix*. The live non-attenuated vaccine is a live oocyst vaccine isolated from chickens, prepared from anticoccidial sensitive strains of *E. acervulina*, *E. maxima*, *E. maxima* MFP, *E. mivati*, and *E. tenella*.

2.2. Experimental design

One hundred and twenty day-old Avian-48 broiler chicks (average body weight 42 g) were randomly divided into 6 groups (G1- G6, 20 chicks/group). Group 1 (G1) was vaccinated with attenuated precocious type vaccine (A) and non-challenged. G2 was vaccinated with non-attenuated wild-type vaccine (B) and non-challenged. G3 was non-vaccinated non-challenged negative control. G4 was vaccinated with attenuated precocious type vaccine (A) and challenged with *E. tenella*. G5 was vaccinated with a non-attenuated wild-type vaccine (B) and challenged with *E. tenella*. G6 (positive control group) was non-vaccinated challenged with *E. tenella* (NVC). Before allocation of chicks, all pens were cleaned using water and soap, then disinfected using ammonia releasing compound to release ammonia for the destruction of the coccidial oocyst. All pens were heated using an electric heater in order to maintain the temperature within 25-30°C during the whole period of the experiment. Also, all pens were well-lightened electrically. Chicks in groups 1, 2, 4, and 5 were vaccinated via eye dropping at day-old (every 100 chicks = 1ml). All vaccinated groups were challenged by inoculation of 10⁵ sporulated oocysts of *E. tenella* (Messai et al., 2014). Sporulated oocyst of *E. tenella* isolate was kindly obtained from the department of parasitology, Animal Health Research Institute, Tanta Branch. The isolate was propagated from a single *E. tenella* oocyst in

coccidia free broiler chick. Before infection, fecal samples from G3 were examined microscopically and cecal specimens were examined histopathologically to confirm the absence of coccidial infection. From the 5th day post-infection, fecal samples from all vaccinated groups (G1, G2, G4, and G5) were also examined to confirm the presence of oocysts. All the experimental chicks were vaccinated against ND, IB, and IBD in accordance with the local vaccination program. Two hundred and ten fecal samples were collected (five samples from each pen daily from the 7th day post-infection till the 14th day) from all groups for counting of the oocysts. Three chicks from each group were randomly collected and sacrificed for examination of cecal lesions, cecal mucosal scraping, and recording of lesion scores on the 7th day post-infection. Cecal tissues specimens were collected for the recording of histopathological lesions. Freshly voided droppings were collected daily from each experimental group (5 samples from each group) and were preserved in potassium dichromate 2.5 % till examination by McMaster slide. On weekly basis, each group was weighed individually starting from the 28th day till two weeks post-challenge. Mean body weight, average body weight gain, feed conversion rate (FCR), and feed consumption were calculated as follows:

Mean body weight

$$= \frac{\text{gross live weight of birds}}{\text{total number of birds}}$$

Average body gain per bird

$$= \frac{\text{average final weight of live birds in a pen} - \text{average initial weight of all birds in that pen}}{\text{total feed consumed}}$$

$$= \frac{\text{total feed consumed}}{\text{total number of birds}}$$

$$\text{Feed conversion rate} = \frac{\text{feed consumption}}{\text{average weight gain}}$$

2.3. Oocyst count

Oocyst count was done by collecting 3 fecal samples from each group daily from the 7th day to the 13th day post-infection. The oocyst per gram (OPG) was counted using McMaster counting technique according to (Haug et al., 2006). OPG feces = number of oocysts in 2 chambers x 50.

2.4. Post-mortem examination and lesion score

Post-mortem examination of sacrificed and freshly dead birds was carried out to record cecal coccidiosis lesions (Johnson and Reid, 1970). Cecal lesion score was used to evaluate of the efficacy anti-coccidial vaccine at the 7th day post-infection (Johnson and Reid, 1970). The severity of *E. tenella* infestation lesions is usually proportional to the number of oocysts ingested by the bird and typically correlates with other parameters such as weight loss and droppings scores of experimentally examined chicks. A score of 0-4 is assigned to a bird where 0 is normal (no gross lesion) and 4 is (the most severe gross lesion) (Johnson and Reid, 1970). It was carried out as presented in Table 1.

2.5. Histopathological examination

Histopathological cecal specimens were collected on the 7th day after the challenge and fixed in 10% formalin, dehydrated in graded alcohol concentration, cleared with xylene, and embedded in paraffin. Embedded tissues were sectioned at 5 µm thickness and stained with hematoxylin and eosin (H&E) and finally examined microscopically (Lillie and Fulman, 1976).

Table (1): The lesion scores of *E. tenella* infestation

Score	Lesion
1	Few reddish petechiae in cecal serosa. Brownish cecal contents, no thickening of the cecal wall. Few reddish petechiae on the cecal mucosa.
2	Numerous serosal petechiae. Bleeding in the mucosal surface. Slight thickening of the cecal wall.
3	Coalesced petechiae on the serosal surface of the cecum. Severe bleeding and sloughed mucosal surface. Clotting appearance in the distal end of the cecum. Marked thickened cecal wall.
4	Cecal core with whitish castes and absence of normal cecal contents. Marked thickened cecal wall. Gangrene and rupture of the cecal wall.

2.6. Statistical analysis

Statistical analysis was performed using SPSS 23.0. The significant difference among different chickens was carried out using one-way ANOVA followed by Tukey's adjustment test. The significant difference between the mean numbers of oocysts among different examined groups at different time points was carried out using two-way ANOVA. Data were presented as mean±SEM and the significance was declared at $p \leq 0.05$.

3. Results

3.1. Feed conversion rate

In the period of 28 – 35 days, the FCR did not change significantly between the first 3 groups, while G4 and G5 were significantly higher than other groups (Table 2). In the period of 35 – 42 days, G3 showed significantly lower FCR while G6 showed significantly higher FCR than other groups. The FCR for the whole period between 28 – 42 days was highest in G6 and lowest in G3. The FCR was significantly higher in G4 and G5 than G1 and G2.

Table (2): The feed conversion ratio in all groups at different timepoints

Groups	FCR (28 – 35 days)	FCR (35 – 42 days)	FCR (28 – 42 days)
G1	1.65±0.06 ^c	2.38± 0.10 ^b	1.97±0.14 ^c
G2	1.75±0.07 ^c	2.43±0.11 ^b	2.04±0.15 ^c
G3	1.50±0.06 ^c	1.65±0.07 ^c	1.55±0.11 ^d
G4	1.94±0.07 ^b	2.52±0.10 ^b	2.20±0.16 ^b
G5	1.97±0.08 ^b	2.62±0.11 ^b	2.26±0.15 ^b
G6	2.63±0.11 ^a	3.24±0.17 ^a	2.90±0.42 ^a

Treatments within the same column with different superscript letters show significant differences ($P < 0.05$).

3.2. Body weight gain

The BWG for the period between 28 – 35 days was highest in G3 and lowest in G6, but it did not change significantly among other groups (Table 3). In the period of 35 – 42 days, only G3 showed significantly higher BWG than all other groups. The BWG for the whole period between 28 – 42 days was highest in G3 and lowest in G6. However, no significant difference was noticed among other groups.

3.3. Oocyst count

There was no significant difference among the first 4 groups across time, while G5 and G6 had a significantly higher oocyst count at all times of measurements than other groups (Table 4). Within groups,

there was no significant difference in the first 3 groups across time. On the other hand, G4-G6 showed a gradual increase in oocyst count by time until the maximum significant increase on day 4 of the challenge followed by a significant decrease in the following days.

Table (3): BWG (g) in different groups at different timepoints

Groups	BWG (28 – 35 days)	BWG (35 – 42 days)	BWG (28 – 42 days)
G1	743.75±32.84 ^b	644.69±45.25 ^a	1388.44±97.00 ^b
G2	748.33±34.22 ^b	632.67±58.10 ^a	1381.00±111.18 ^b
G3	872.81±42.66 ^a	844.69±37.80 ^a	1717.50±131.20 ^a
G4	734.69±35.58 ^b	630.31±48.01 ^b	1365.00±96.33 ^b
G5	686.25±27.89 ^b	628.13±56.70 ^b	1314.38±82.38 ^b
G6	579.29±24.80 ^c	529.29±74.19 ^b	820.71±161.10 ^c

Data were presented as mean± SEM. Values in the same columns with different letters (a - c) differ significantly at $P \leq 0.05$.

3.4. Lesion score, oocyst index, and mortality rate

Challenge with 10^5 sporulated oocyst of *E.tenella* resulted in a mortality rate of 10 % in G-6 mortality on the 8th and 9th day post-challenge (36 and 37-day old) but there was no mortality in other groups (Table 5). The lesion score was significantly higher in G6 followed by G5 and then G4 than other groups. The oocyst index of vaccinated challenged groups was lower than that of the non-vaccinated challenged group (G6).

3.5. Histopathological finding

G1 and G3 showed a normal histological structure of cecal mucosa, submucosa, tunica muscularis, and serosa. The enterocytes and mucosal crypt epithelium appeared normal and free from various developmental stages and oocysts of *Eimeria* Spp. (Fig. 1A, B). In contrast, G2 showed multifocal interstitial leucocytic cellular infiltration mainly lymphocytes and few heterophils in the lamina propria of the intestinal mucosa. Diffusely, large areas of the intestinal villi and mucosal crypt epithelium appeared normal and free from developmental stages and oocysts of *E. tenella* (Fig. 1C).

G46 showed circumferential expanding the lamina propria of the cecum and infiltrating the mucosal/crypt epithelium were myriad developing coccidial life stages, including numerous intracellular macrogamonts and microgamonts, and both intra and extracellular schizonts; and many developing oocysts which comprise the majority of the developing stages (Fig. 2D-F). Macrogamonts were round in shape with a single, central nucleus and a peripheral ring of eosinophilic granules. Multifocally, the mucosa is eroded with loss of enterocytes and replacement by hemorrhage, fibrin, eosinophilic cellular and karyorrhectic debris, and inflammatory cells. Expanding the lamina propria and submucosa was a moderate cellular infiltrate composed of lymphocytes, macrophages, and few heterophils admixed with fibrin and edema (Fig. 2D-F).

In G4, the mucosal/crypt epithelium of the cecum appeared normal and free from developmental stages and oocysts of *E. tenella*. Occasionally, the columnar epithelial cells of few crypts of Lieberkuhn showed mild hyperplasia and exhibit infection with gametocytes, and oocysts of *E. tenella*. The remaining layers were normal (Fig. 2A, B). In G5, the cecum showed marked histopathological changes similar to G6 (Fig. 2C-F).

Table (4): The mean number of oocysts among the different groups

Groups	Days post-infection						
	7 th	8 th	9 th	10 th	11 th	12 th	13 th
G1 ^a	1370±309	400± 132	350± 242	20± 45	460± 373	120±144	10±22
G2 ^a	450± 187	260± 96	170± 189	60± 65	340± 446	100±173	110±219
G3 ^a	0	0	0	0	0	0	0
G4 ^a	1640± 879 ^c	3520± 1035 ^c	7680± 2175 ^c	12680±1919 ^d	3090± 738 ^c	2660±1204 ^c	530±115 ^e
G5 ^b	3700± 1037 ^c	12920± 1588 ^c	25250±3287 ^d	19000±886 ^d	5000±816 ^c	2800±517 ^c	940±89 ^e
G6 ^b	86050± 42966 ^c	107450± 53722 ^d	131120±10633 ^d	109340±9892 ^e	67800±4232 ^c	39280±4118 ^f	19924±793 ^g

Treatments within the same column or different rows with the same superscript letters show no significant differences ($P < 0.05$)

Table (5): Lesion scores, oocyst index, and mortality %

Group	Lesion score	Oocyst index	Mortality %
1	1	+	0
2	1	+	0
3	0	0	0
4	1.67	+	0
5	2.67	++	0
6	3.67	+++++	10

Oocyst index using lens 10x (Hilbrich, 1978):

- 0: no oocyst / field.
- +1: 1-10 oocysts / field.
- +2: 11-20 oocysts / field.
- +3: 21-50 oocysts / field.
- +4: 51-100 oocysts / field
- +5: > 100 oocysts / field

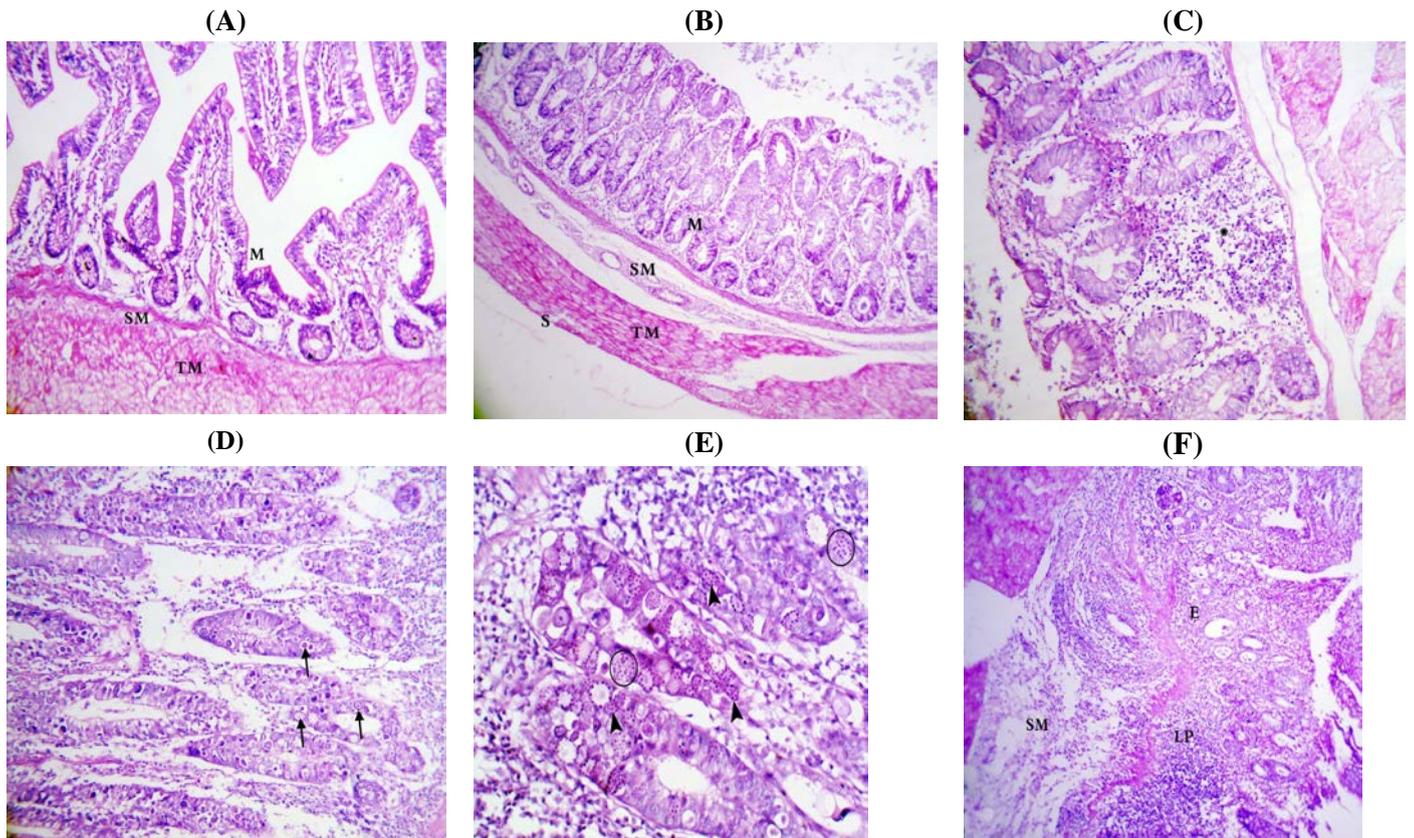


Fig.1. (A) The cecum of the control chick (G3), x 200. (B) The cecum of vaccine A administrated chick (G1), x 100. (C) The cecum of vaccine B administrated chick (G2) showing interstitial lymphocytic cellular infiltration (asterisk) in the lamina propria of the mucosa, x 200. (D-F) The cecum of chick infected with acute coccidiosis on 28 days (G6), showing (D) marked infection of the columnar epithelium of crypts of Lieberkuhn with myriad developing coccidial life stages (arrow), x 200; (E) hypertrophied crypt epithelium with developing coccidial life stages; microgamonts (arrowhead), schizonts (circle) with numerous basophilic merozoites, x 400; and (F) mucosal erosion (E), x 100. H&E stain. LP, lamina propria; M, mucosa; S, serosa; SM, submucosa; TM, tunica muscularis.

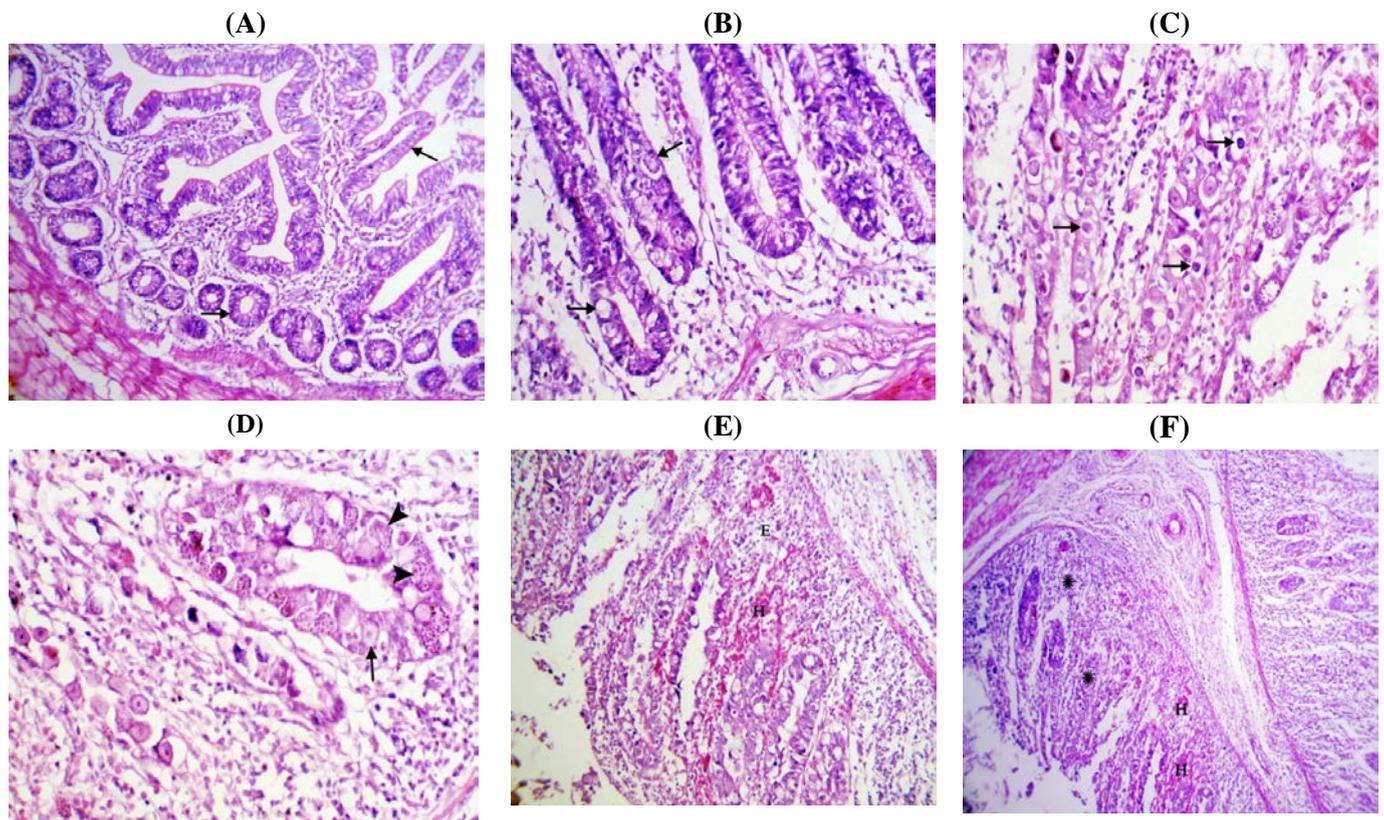


Fig. 2. (A, B) The cecum of vaccine A administrated chick infected with acute coccidiosis (G4), showing (A) normal mucosal/crypt epithelium (arrow) free from developmental stages and oocysts, x 200; (B) infection of the columnar epithelial cells (arrow) of few crypts with gametocytes and oocysts, x 400. (C-F) the cecum of vaccine B administrated chick infected with acute coccidiosis (G5), showing (C) numerous developmental stages and oocysts (arrows) within enterocyte cytoplasm, x 400; (D) marked infection of the crypt epithelium with gametocytes (arrow) and schizonts (arrowheads), x 400. (E) mucosal erosion (E) with loss of enterocytes and replacement by hemorrhage (H), x 200; and (F) damage of cecal glands with leucocytic cellular infiltration (asterisk), and hemorrhages (H), x 100. H&E stain.

4. Discussion

In the present study, the efficacy of live attenuated (precocious) anti-coccidial vaccine was evaluated in comparison with live non attenuated (wild) anti-coccidial vaccine which. The effect of coccidiosis on vaccinated and non-vaccinated chickens was also compared. In general, we found that vaccinated challenged groups (G4 and G5) give a high improvement in FCR and BWG when compared with the non-vaccinated infected group (G6).

Lower FCR indicated higher body weight gain. The coccidial infection causes depression, diarrhea, and loss of appetite which could lead to bodyweight loss. There was no noticeable difference in FCR between G4 and G5, but these two groups showed a significantly lower FCR than G6. In agreement, Abdel-Aziz (2011) also found that Coccivac-B® vaccinated group exhibited lower FCR than the non-vaccinated group. These results also agreed with Rafiqi et al., (2017) who reported a significant increase in relative weight gain and improved FCR following immunization of birds at days 7 and 21 of age with 1000 live sporulated oocyst of *E. tenella* and challenged with the homologous strain of parasite on day28 of age. Another point of agreement is that in a commercial trial in South America comparing Livacox® with prophylactic medication (unspecified), Livacox® vaccinated birds had a higher FCR but lower live weights than the medicated controls (Shirley and Bedrnik, 1997). The vaccinated birds with Coccivac B® vaccine had the best FCR, as revealed in a study for comparison of salinomycin and diclazuril efficacy and Coccivac B® (Hamed E.M., 2011). Furthermore, our data are compatible with those of Waldenstedt et al., (1999), who found that vaccinated birds had a lower FCR than

those medicated with narasin. In contrast, vaccinated flocks had higher FCR than non-vaccinated but medicated flocks (Williams et al., 1999).

Our findings in consistence with Hamed, (2011) and Rafiqi et al., (2017) showed a significant increase in BWG in the vaccinated challenged groups (G4 and G5) as compared to the non-vaccinated challenged group (G6). Additionally, BWG in the vaccinated community was greater than that in the medicated and non-vaccinated groups (Danforth et al., 1998; Norton et al., 1989). However, Ruiz and Tamasaukas, (1995) did not find a weight difference between vaccinated birds and non-vaccinated groups.

The vaccinated challenged with the attenuated vaccine (G4) showed a significant decrease in the oocyst shedding as compared to the vaccinated challenged with the non-attenuated vaccine (G5). In agreement, Abdel-Azize (2011) and Ruiz and Tamasaukas (1995) also found that chicks vaccinated with Coccivac vaccine and challenged with oocysts showed a significant reduction of oocyst count in comparison with the non-vaccinated challenged group. Similarly, Rafiqi et al., (2017) reported that immunization with live oocysts of *E. tenella* resulted in a significant reduction in oocyst output (93.74%) indicating that oral immunization of chickens against *E. tenella* was effective in preventing the clinical disease and decreasing the oocyst burden in poultry farms. In parallel, G4 also had a reduced oocyst index and fecal score than other groups.

In the present study, the lesion score significantly decreased in G4 and G5 than G6. This result agrees with Abdel-Azize, (2011); Ruiz and Tamasaukas, (1995) who reported dramatically decreased clinical symptoms, dropping scores, mortality, and cecal lesion scores for *E. tenella* infection in vaccinated birds as compared to the control (non-

vaccinated) group. Our results also agree with Bushell et al., (1992) who reported that, in flocks vaccinated with Paraocx® vaccine, coccidiosis was not detected with no lesions appeared in the post-mortem examination. In support, administration of Fortegra® (a coccidial vaccine including precocious and classic strains of *E. maxima*) resulted in lower lesion scores at 14, 17, and 21 days post-vaccination (Madison, 2015).

Our results agreed with Soomro et al., (2001) who found that histopathological lesions of cecal coccidiosis were loss of epithelial tissue, congestion of blood vessels, followed by leakage of blood, severe muscular edema, necrosis of submucosa, loss of villi, cluster of oocysts and marked hemorrhage, necrosis of cecal mucosa and lymphoid cells hyperplasia that all appear in G6. The histopathological changes in G5 were similar to those in G6. However, G4 showed no histopathological changes and showed normal intestines as chickens in G3. Similarly, Saravanan et al., (2014) found a destruction of cecal epithelium in all groups administered 10 and 20µg of live sporozoite antigen and Sharma et al., (2015) found coccidial oocysts in the intestine's lamina propria and the epithelial cells of the caecum's submucosal glands, massive infiltration of heterophils and mononuclear cells, together with desquamation and sloughing of enterocytes in the intestine resulted in intestinal villi necrosis, in addition to submucosal blood vessel congestion, fibrosis, and edema, as well as submucosal degeneration. Increased FCR and decreased BWG in G6 may be due to the presence of cecal lesion caused by *E. tenella*, which destroys the absorptive mucosal surface of the intestine (Logan et al., 1993; Kheirabadi et al., 2008; Marien and Gussem, 2007; Mathis et al., 2018).

There are marked differences between the attenuated vaccines and the non-attenuated vaccine concerning precocity, fecundity, and pathogenicity. The non-attenuated vaccine induced higher lesion scores and induced greater oocyst output compared to the attenuated vaccines (Mathis et al., 2018). In agreement, we also reported that the usage of anti-coccidial vaccine has significant protective efficacies in broilers with great potential with attenuated strain vaccine (precocious strains).

Conclusion

Administration of coccidial vaccines improved BWG and FCR and decreased oocyst shedding, lesion score, bloody dropping, and mortality rates with best effect for the live attenuated vaccine in chickens.

Conflict of interest

The authors declare that they have no conflict of interest.

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