NIGERIAN AGRICULTURAL JOURNAL
ISSN: 0300-368X
Volume 54 Number 2, December 2023 Pg. 310-323
Available online at: http://www.ajol.info/index.php/naj
https://www.naj.asn.org.ng

Pathology, Haematology and Biochemistry of Coccidiosis in Broiler Chickens

[1]Ukonu, C.E., [2]Ogo, I. N., [3]Daniel-Igwe, G., [1]Innocent, I.N. and [1]Okereke, C.O.

[1]National Root Crops Research Institute, Umudike
[2]National Veterinary Research Institute, Vom
[3]Michael Okpara University of Agriculture, Umudike.
Corresponding author's email: erastusdavid4real@gmail.com

Abstract
Coccidiosis is the most economically important parasitic disease of poultry. Forty five day-old broiler chicks were used for this study. They were divided randomly into three equal groups (Groups A to C). Each group comprised 15 chicks with three replicates in each group. The groups were arranged as follows: Group A: Control (uninfected - untreated), Group B: Infected - untreated and Group C: Infected - treated. The feed intake in the two infected groups decreased but it was more severe in the Group B than in Group C by day 7 PI. By days 14 to 42 PI the two infected groups competed favourably and were higher in feed intake when compared to the control birds. But their weight gain were far below the control until the end of the experiment, this is due to the pathological changes the disease organism caused and was demonstrated by histopathological studies of the slide. The disease and its effect was severe in the group B. other parameters were in agreement that the effect was severe in group B and less severe in group C. Hence, coccidiosis negatively affected both the activity score and faecal score in the groups B and C. Coccidiosis presented clinical signs and pathologic changes in the affected birds and these affect profitability of the rural poultry farmers / industry. Hence, the disease is a real problem in the poultry industry and the gain of confronting the challenge posed by it is worth the investment.

Introduction

The challenge posed by coccidiosis in the poultry industry is enormous. For successful control of coccidiosis to be attained, there is need for continuous research geared towards proffering solutions. This is because as the years go, the emergence of the resistant strains of *Eimeria* organisms have become increasingly worrisome. The events of the past decades have shown that the causative organisms have not remained where they were. Field experiences have shown that the organisms not only develop resistance faster but also there has been a shift in age at which broiler chickens can be affected with coccidiosis. Chickens now are affected in the first week of life (Okonkwo *et al.,* 2019). Poultry industry is concerned with the production, processing, and marketing of poultry and poultry by–products. Poultry production is one of the rapidly growing industries in Nigeria. It is also a source of protein hence very useful in meeting protein demands in our ever-increasing population (Abdu and Musa, 2014). Coccidiosis in poultry has been known for many years, it is still considered the most economically important parasitic condition affecting poultry production worldwide. The aetiologic agents are of the Genus *Eimeria* with up to nine different species found to affect chicken (Degussein, 2007). *Eimeria* is with few exceptions intracellular parasites of the epithelial cells of the intestine. Each species has a single host in which they undergo asexual and sexual multiplication. In practice, faecal examination for oocysts has been a common method of diagnosis. (Soulsby, 1982 and Fakae, 1987). The diagnosis of coccidiosis is based on clinical signs, scatology and pathomorphological and pathohistological analysis (Long and Joyner, 1984, Conway and McKenzie, 2007). In recent years, various biochemical and molecular methods have also been used for diagnosis (Morris and Gasser, 2006). Serology is the predominant method of monitoring coccidiosis in commercial poultry, examination of blood smears, bone marrow and clinical chemistry values is rarely done. The specific diagnosis of infection plays a key role in the prevention, surveillance and control of coccidiosis (Wakenell, 2010). The *Eimeria* experimental infection will provide comparative information on the haematology, biochemistry and histopathology of infected and non-infected broiler chickens.

Materials and Methods

Study location

The study was carried out at the Michael Okpara

University of Agriculture, Umudike (MOUAU) in the Department of Veterinary Pathology, College of Veterinary Medicine. MOUAU is located between 5°28`33" N and 7° 32`56" E (Anonymous, 2014). The study design included haematological, biochemical and pathological studies. The study was conducted between December 2019 and March 2020 for the *Eimeria* experimental infection.

Experimental drug

Amprolium (Amprolium 250WSP®) a commercially available anticoccidial drug for the routine treatment of avian coccidiosis (due to *Eimeria* species) was used, this was because amprolium gave a better result in a previously concluded experiment of all the drugs used.

Eimeria species strains

The sporulated oocysts used to infect the animals were obtained from the Parasitology Department of the National Veterinary Research Institute, Vom Jos. Plateau State. The collected field strains of sporulated *Eimeria tenella* oocysts were packaged and preserved in an ice pack and transported to the Department of Pathology, College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike Abia State. On arrival, the sample was confirmed to contain sporulated *Eimeria* oocysts with the light microscope and then stored in the refrigerator at 4°C and was used within two weeks for the studies.

Study population

Forty-five (45) day-old Ross 308 broiler unsexed chicks, obtained from a local commercial broiler hatchery were used for the experiment. The birds were kept under good hygienic conditions. Drinking water and feed were offered *ad libitum*. Chicks were fed on a balanced commercial ration free from anticoccidials. The chicks were floor-reared in separate units throughout the experimental period. They were routinely vaccinated against Newcastle and infectious bursal diseases.

Experimental design

A total of forty-five-day-old Ross 308 broiler chicks were divided randomly into three equal groups (from Groups A to C) and weighed by a restricted randomized procedure (RRP) that initially equalised their weights. Each group comprised 15 chicks with three replicates in each group and each replicate had five chicks. The groups were arranged as follows:

Group A: Control (Uninfected - untreated).
Group B: Infected - untreated.
Group C: Infected - treated (with Amprolium).

Birds in groups B and C were infected with an equal dose of 10,000 sporulated *Eimeria tenella* oocysts per bird *per os* at the 14th day of life. Treatment by the drug was started in the drinking water according to a recommendation by the manufacturer on the 6th day post-infection (20th day of age) or following the appearance of oocysts in faecal samples (oocysts were detected in the faeces of chickens by day 5 PI and the treatment started the next day; 6th day PI).

Clinical examination

Clinical signs such as depression were checked by activity score, and diarrhoea was checked by faecal score and recorded. The severity of diarrhoea (faecal score); a scale of 1 – 5 was used to measure normal faeces and the degree of deviation from normal) as follows: 5 – no diarrhoea, 4 – mild, 3 – moderate, 2 – marked and 1 – more marked diarrhoea. A similar scale was used for the activity score.

Parasitological examination: oocyst counts

Fresh faecal droppings were collected randomly from the litter spread on the ground from each group for daily oocyst counts from the 4th to 19th-day post-infection (dpi) until when only a few or no oocyst could be detected in faecal droppings (related to oocysts shedding curve of the control group). The mean number of oocysts per gram of faeces for each group was counted and for the large counts, the McMaster counting technique according to the method described by Long *et al* (1976) was used.

Weight gain/feed intake

Three birds were randomly picked from each group and weighed weekly from zero-day of infection till day 42 PI. The body weights were recorded, calculated and expressed in grams and average. Also, feed intake was determined by a measured daily feed allowance minus the total feed leftover per group per week.

Haematology

Blood samples from each animal were collected weekly from the jugular vein of 3 chickens randomly selected from each group using a 2 ml sterile syringe, a 23-gauge needle and cotton wool with methylated spirit. Each blood sample was transferred immediately into a sterile tube containing the anticoagulant, ethylenediaminetetraacetic acid. Red blood cell (RBC) and total white blood cell (WBC) counts were determined by the haemocytometer method. PCV was determined by the microhaematocrit centrifugation method. The haemoglobin concentration was determined by the cyanomethaemoglobin method (Kachmar, 1970).

Biochemical analyses

The Microhematocrit tube was filled with EDTA-anticoagulated blood using capillary action. The tube was then sealed, placed in a microhematocrit centrifuge, centrifuged for 5 minutes and then removed from the centrifuge. The microhaematocrit tube was marked above the buffy coat, using a diamond pen. The tube was then cleanly broken at the marked line. The end of the tube containing plasma was then placed at the interface of the refractometer lid and the plasma was transferred to the window by capillary action. The refractometer plate was then closed on top of the plasma and the interface between the light and dark regions was identified through the visualization window and the reading of plasma protein was taken according to George (2001). Fibrinogen was determined according to Lawrie *et al*. (1998).

Post-mortem examination and histopathology

Necropsy was done on all birds that died during the experiment. Also from day 10 PI, 3 birds per group were euthanatized at varied intervals till the end of the experiment for necropsy and histopathology. Gross Lesions were determined by macroscopic examination of the intestine and caeca of each bird using Elbahy *et al.* (2006) as a guide.

Statistical analysis

The data obtained from this study were presented as means ± SEM (standard error of means) and were statistically analysed using analysis of variance (ANOVA). A comparison of groups was performed using the least significant difference (LSD) at $P < 0.05$ according to Petrie and Watson (1999). Other results were presented in plates.

Results and Discussion

Feed intake in broiler chickens

The weekly feed intake in broiler chickens is presented in Table 1. By the 7 days PI (14 – 21 days of the chickens' live) the feed intake of Group B and Group C were 390.5±3.6g and 419.4±19.7g respectively, which were significantly lower ($p<0.05$) than that of the control (518.9±7.8g). By day 14 PI, the feed intake by groups B and C had increased with no significant difference between their feed intake and those of the control group. By day 21 PI, the feed intake of groups B (759.1±7.3g) and C (690.8±41.5g) rose significantly above the feed intake of the control (596.3±8.5g). This trend on day 21 PI was similar to that of day 28 PI. By days 35 and 42 PI, feed intakes of the three groups were essentially similar. The total feed intake per broiler during the experiment was similar in the three groups. The feed intake in the two infected groups was reduced but it was more severe in group B than in group C treated by day 7 PI. This agrees with Anosa *et al*., (2011) and Okonkwo *et al.,* (2019) who observed that coccidiosis causes reduced appetite. Unlike their findings, by days 14 to 42 PI the two infected groups (B and C) competed favourably and were higher in feed intake when compared to the control group. Cumulatively, however, the average total feed intake value of groups B (4472.1±70.6g), and C (4483.7±55.8g) when compared to the control group A (4425.5±160.7g) were similar due to increased feed intake of the infected groups post-treatment and post-recovery. This is thought to be part of the inherent recovery mechanism in which infected birds attempt to eat more post-treatment. These increased feed consumption by day 21 PI in groups B and C birds were not directly proportional to the increase in weight gain of the birds in the groups compared to the control by day 21 PI.

Mean body weights of broiler

The mean body weights of broiler chickens are presented in Table 2. Before infection, the weights of birds in the three groups were essentially similar. By day 7 PI the growth rates of the infected and infected treated groups were significantly lower ($P< 0.05$) than the controls. This persisted on days 14, 21, 35 and 42 PI as groups B and C continued to grow at a significantly lower rate than the birds in the control group. The weight of the birds in the three groups before infection were similar. Following infection, the two infected groups B and C weight gain rose at a significantly lower rate till the end of the experiment compared to the control group A birds despite the increased feed consumption in groups B and C birds from days 14 to 42 PI. This was more so in group B. The reduction in the weight gain in groups B and C was presumably due to damages that the *Eimeria* developmental stages caused in the intestinal tract of the infected birds which negatively affected efficient digestion and absorption of the feed consumed by the birds; furthermore diarrhoea and blood loss presumably exacerbated the situation. This agrees with the findings of Okonkwo *et al.* (2019), who reported that coccidiosis hurts weight gain in growing birds. Expectedly, the growth rate of the group C was better than that of the group B. This shows that treatment reduces the coccidia infection and improves the growth rate. However, the earlier weight losses due to infection were not reversed completely during the period of the study.

The Activity Score

The activity scores of broiler chickens are presented in Table 3. By day 0 PI, the activity of birds across the groups was similar. By day 4 PI, the two infected groups became significantly less active than the control. Reduced activities were more severe from day 4 PI to day 11 PI in the infected groups but were generally more marked with group B than group C. Thereafter from day 12 PI, activity scores improved in the two infected groups, particularly in group C in which activity returned to normal by 17 PI.

The results of the activity score have shown that coccidiosis reduces the activity of the infected birds significantly unlike the control group. The activity score for the group B birds was significantly lower than the other groups. This is in agreement with previous studies on the activity of birds infected with *Eimeria* following treatment with amprolium (Campbell and Coles, 1986; Okonkwo *et al.,* 2019). The use of amprolium demonstrates the positive effect of drugs in improving the activity score or survivability of group C.

Faecal scores of broiler chickens

The faecal scores of broiler chickens are presented in Table 4. By day 0 PI, the faecal scores across the groups were similar. By day 4 PI, the faecal scores of groups B and C became significantly lower than the control group. The faecal scores of the infected groups were markedly low from day 4 PI to day 11 PI but it was generally more marked with group B than group C. Their faecal scores improved from day 12 PI, in the two infected groups, particularly in the infected treated group in which faecal score returned to normal by day 15 PI. The faecal score (brown faecal material) of the experimental birds showed a correlation between the activity score and faecal score of groups B and C. The two groups were significantly lower than the control group. Like in the activity score, a faecal score of groups B and C were low,

more severe and marked in group B than in group C. Campbell and Coles (1986) and Okonkwo *et al*. (2019), stated that faecal scores are used as part of indicators of morbidity in addition to other parameters. From day 12 PI, faecal score improvement in the two groups further confirms that coccidiosis is self–limiting (Fakae, 1987; Anosa *et al*., 2011; Okonkwo *et al.,* 2019).

Plasma protein and fibrinogen

The plasma protein and fibrinogen levels of broiler chThe plasma are presented in Table 5. Plasma proteins of the three groups pre-infection were similar. By day 3 PI the plasma protein of groups A and C were similar and significantly higher than group B. By day 7 PI, groups A and B were similar but significantly higher than group C. By day 10 PI, groups B and C were similar but were significantly higher than group A. By day 14 PI, groups B and C were similar and were significantly lower than group A. By days 17, 21, 24, 28, 35 and 42 PI, the plasmwasotein was similar in the three groups. By days 0, 7, 10, 21, 24, 28, 35 and 42 PI, the fibrinogen levels are similar in the three groups. By day 14 PI groups A and B were similar but group A was significantly higher than group C birds. Groups B and C were similar. The fibrinogen values by day 0 PI were similar across the groups but by day 3 PI, the value of groups B and C birds rose sharply and were significantly higher than group A birds. By days 7 and 10 PI, the fibrinogen values were similar statistically across the groups. By day 14 and 17 the fibrinogen values were similar in groups A and B but were significantly lower than group C birds in both days. By days 21, 24, 28, 35 and 42 PI, the values across the groups were similar statistically. By day 3 PI the plasma protein of group B dropped significantly when compared to birds in groups A and C. This is thought to be due to haemorrhage and fluid loss into the intestinal lumen and this is in agreement with earlier workers who observed that coccidiosis causes a reduction in plasma protein levels. (Schildt and Herrick, 1955; Stephens, 1965; Preston – Mafham and Sykes, 1967; Chapman *et al.,* 1982; William *et al,* 1985). However, the reduction in blood plasma proteins was short-lived as the values in the three groups were similar by days 7 and 10 PI. This could be due to the immediate recovery mechanism from the disease challenge. However, by days 14 and 17 the reason for the significant decrease of plasma proteins of groups A and B below the group C birds is uncertain and may need further investigation. By day 21 to 42 PI, the plasma proteins of birds across the groups were similar. It could be concluded that recovery from plasma protein loss is rapid following coccidial disease. This agrees with Chapman (2007). It is important to say that because of the rapid recovery shown by the plasma protein in this study, plasma protein measurement may not be a good indicator of the adverse effect of coccidiosis in broiler chickens. The fibrinogen concentrations of the birds across the groups by days 7, 10, 21 – 42 were similar. The fibrinogen picture did not truly reflect the pathologic effects of coccidiosis on the chickens affected and so may not be a good indicator for monitoring coccidial adverse effects.

Haematology

The erythrocyte and total white blood cell counts of broiler chickens are presented in Table 4.8. The erythrocyte values of the birds in the three groups were similar initially but following infection, groups infected and infected-treated birds showed anaemia when compared to the control birds. However, from day 14 to 42 PI the Group B birds progressively recovered from the anaemic state. Furthermore, the rate of recovery was faster in the Group B birds but the erythrocyte values of the control birds were better than the two infected groups at the end of the study. The blood cells (WBC) of groups B and C by days 0 and 35 PI were similar but were significantly lower than the control. By days 3, 7, and 42 their WBCs were similar. By days 10 and 14 PI groups A (control) and C values were similar and significantly higher ($P < 0.05$) than group B birds. By days 21 and 28 PI, groups A and C were similar and were significantly lower ($P < 0.05$) than group B birds. The erythrocyte values of the birds in the three groups were similar initially but following infection, groups B and C birds showed anaemia when compared to the control birds. However, from day 14 to 42 PI the groups B and C birds progressively recovered from the anaemic state. The erythrocyte findings agree with earlier studies (McDougald and Fitz–Coy, 2008; Anosa *et al.*, 2011; Chaiwat *et al*., 2013; Okonkwo *et al.,* 2019). Furthermore, the rate of recovery was faster in the infected–treated birds. The total white blood cell changes in the control and infected birds were inconsistent during the experiment with no pattern. Total WBC values for the infected birds (group B) were, however, significantly higher than in the control on day 2 and 28 PI; this is consistent with the reports of Chivaramaiah *et al*. (2014). This was presumably due to the intestinal tissue damage caused by the coccidia in the birds and tissue response.

Experimental Oocyst count

The mean oocyst counts of experimental broiler chickens are presented in Table 7. At days 0-4 PI, there was no detectable oocyst in the faecal samples of the three groups. The control had no oocysts throughout the experiment. Group B showed oocysts in their faeces by day 5 PI (384.0±5.0/Opg), and the oocysts increased with time, reaching the highest level of 2869.3±202.6/Opg by day 18 PI. However, oocyst counts dropped to 1696.3±207.4 by day 19 PI. Oocyst counts were similar in groups B and C birds, although they were slightly higher in group C birds. Following treatment with amprolium on day 7 PI, oocyst counts in group C declined to 1453.3±50.0/Opg on day 13 and to 618.0±463.5/Opg on day 19 PI. The presence of detectable *Eimeria* oocysts in the faeces of birds by day 5 PI in groups B and C birds agrees with the report of Ngongeh *et al*. (2017), who detected oocysts in the faeces of infected birds by day 4 PI. The oocyst count continued to rise until day 18 PI in group B, peaked at day 18 PI and declined afterwards. This agrees with Ngongeh *et al*. (2017), who stated that coccidial infection is self–limiting. In Group C, oocyst count peaked by day 8 PI thereafter, declined to 618.0±4663.5

by day 19 PI. This implies that the use of anticoccidial is very helpful in eliminating the coccidiosis. This is in agreement with other studies (McDougald and Fitz–Coy, 2008; Anosa *et al.*, 2011; Ngongeh *et al*., 2017; Okonkwo *et al*., 2019). The oocyst count in groups B and C birds correlated with their weight gain as the higher the oocysts, the lower the rate of weight gained till the end of the experiment. This is consistent with the findings of Ngongeh *et al*. (2017). This implies that the higher the dose of infection the greater the damage in the intestinal mucosa which invariably interferes with the small intestine digestive and absorptive capacity of the nutrients available in the feed, thereby, diminishing the feed conversion efficiency. Efforts, therefore, should be made to keep the infection dose to the barest minimum (Ngongeh *et al*., 2017), if not eliminated. Group C had the coccidial dose level reduced and this may account for the lower negative impacts on them by the disease. However, the reduction in the oocyst counts in group C was surprisingly poor, indicating some degrees of resistance by the coccidial organism to the drug which has frequently been the drug of choice in this experimental environment in recent times.

Clinical Signs and Pathology

The clinical signs in groups B and C include depression, ruffled feathers, weakness, inappetence, diarrhoea, blood in their faeces (Plate 1) and reduced physical activity found in each of the three replicates of groups B and C. The clinical signs persisted longer in group B while improvement was recorded in group C from the time of treatment. The control group maintained normal activity and the caeca at necropsy did not show any visible deviation from the normal caecal structure histologically (Plates 3 and 4) unlike the infected groups (Plates 5, 6, 8). Grossly the lesions found in groups B and C were accumulation of various quantities of blood and caseous necrotic materials in their caeca (Plates 1 – 2) as well as petechiae, thickening and ecchymosis of caecal mucosae. Groups B and C showed progressive recovery clinically and in all the parameters studied from day 14 PI till the end of the experiment (Tables 1 – 12). Histopathology: Caeca; In the control group, the Caeca appeared normal with the mucosa composed of closely arranged straight tubular glands, lined by enterocytes and goblet cells (Plate 3 and 4). In group B, the lining epithelium of the caecal mucosa showed areas of erosions few points of ulceration, and the presence of oocysts and schizonts (Plates 5 - 7). The Caeca of group C showed areas of erosions devoid of ulcers and the presence of oocysts and schizonts (Plates 8 - 9). Oedema, cellular infiltration and haemorrhages (inflammatory processes) were present in the caecal tissues of groups B and C (Plates 5 – 7 and 9). The liver hepatocytes in the control birds were normal with multifocal aggregations of lymphocytes in the periportal area (Plates 10 – 12). In group B, the liver sinuses and blood vessels were severely congested (Plate 13). The multifocal aggregates of lymphocytes were depleted and they were decreased in numbers, and other areas of lymphoid aggregation were mostly hypoplastic (Plate 13). There were a few areas of hepatocyte degeneration around the portal triad and in the centrilobular area. In group C, the liver sinuses and blood vessels were moderately congested. The multifocal aggregates of lymphocytes were also depleted as in group B (Plate 14). The spleen in the three groups (A - C), appeared normal and was covered by a thin connective tissue capsule. The white and red pulp percentages were essentially the same. The white pulp consists of lymphoid and non-lymphoid white blood cells. The red pulp is an open system and contains widely scattered lymphoid and non-lymphoid cells among red blood cells. All these are normal features in the avian spleen. The presence of high numbers of oocyst schizonts and severe tissue damage in the caeca coincided with the lesions of infection and with high oocyst counts in the parasitological examination. This agrees with Chaiwat *et al*. (2013). Groups B and C showed ballooned caeca, with enlargement of caeca pouches distended with clotted blood and necrotic debris of caecal mucosa in the lumen within the first six days of infection (Plate 1 – 2). These were visible from the serosal surface of the caeca as dark petechiation. The control showed normal mucosal architecture. While groups B and C showed erosion of the epithelial lining and oedema by day 6 PI (Plates 5 – 9). Following treatment, in group C, the mucosal architecture showed recovery. There were haemorrhage and heterophilic infiltration of the lamina propria of the caeca. Additionally, there was the presence of developmental stages of *Eimeria spp* in the mucosa of the intestine (Plate 9). There were also mononuclear cell (lymphocytes, plasma cells and macrophages) infiltrations in the caecal mucosa. The damage/pathology seen in the caeca of the infected birds was associated with the maturation of the second-generation schizonts which caused excessive tissue damage, haemorrhage and disruption of caecal glands / mucosal integrity and necrosis of the mucosae and muscular layer. This is associated with bleeding as seen during the study on day 6 PI and the shedding of oocyst from day 5 PI in both groups B and C birds. This is similar to the findings of McDougald and Fitz–Coy (2008); and Ngongeh *et al*. (2017). The disruption of the mucosal integrity and the consequent bleeding was probably responsible for the low PCV witnessed by days 7 and 10 PI in groups B and C birds which were in contrast to the control group. This is in agreement with Ngongeh *et al*. (2017), and Okonkwo *et al*. (2019). The damage and haemorrhage may also be the reason for the drop in the faecal score by day 4 PI in groups B and C birds and reduced feed intake in groups B and C by days 7 and 14 PI. This is reflected in the reduced rate of weight gain in groups B and C birds from days 7 to 42 PI. This agrees with Okonkwo *et al*. (2019); and Anosa *et al*. (2011). However, group C recovered from the disease and associated lesions much earlier than group B, this was reflected in the several parameters studied, and the former gave better results.

Conclusion

Coccidiosis is still a problem in the study area. The

experimentally infected birds presented clinical signs and haematological, gross and histopathological changes in birds with the disease and these affect the profitability of the industry/investment. Hence, the disease is a real problem in the poultry industry and the gain of confronting the challenge posed by it is worth the investment.

Recommendation

Farmers are advised to target prevention of the disease rather than managing the disease when it occurs. This is because if the disease is allowed to occur before treatment money will be spent yet there will be losses and unit cost of production will rise and the profitability of the business will nose dive. It will also cause contamination of the poultry house and also serve as a source of infection to other poultry units near and far.

References

Abdu, P.A. and Musa, U. (2014). Textbook of Avian Medicine. 1st Ed. Zaria, Nigeria: P. A. Ndahi Printing. Pp. 1,15,131 - 133.

Anonymous. (2014). Umudike. http;Wikipedia.org/wiki/umudike. Accessed on 01/03/19 12.15noon.

Anosa, G.N., Obetta, V.C., Okorie-Kanu, C.O. and Eze, J.I. (2011). Comparative efficacy of toltrazuril, amprolium and Embazine fort against mixed *Eimeria* species infection in broilers. *Nigerian Veterinary Journal*, 32(3): 214 - 217.

Augustine, P.C. and Danforth, H.D. (1990). Avian *Eimeria*: Invasion in Foreign Host birds and generation of Partial Immunity against Coccidiosis. *Journal of Avian Diseases*, 34: 196 – 202.

Campbell, T.W. and Coles E.H. (1986). Avian Clinical Pathology, 4th Ed. W. B. Sounders Company, Philadelphia. Pp. 279 – 300.

Chaiwat, B., Meskerem, A., Nirat, G. and Montakan, V. (2013). Hematological, Biochemical and Histopathological Changes Caused by Coccidiosis in Chickens. *Kasetsart Journal of Natural Science*, 47: 238 – 246.

Chapman, H.D., Fernandes, D.L. and Davison, T.F. (1982). A comparison of the effects of infection with *Eimeria maxima* and dietary restriction on weight gain, plasma metabolites and liver glycogen in the immature fowl, *Gallus domesticus*. *Journal of Parasitology*, 84:205 – 213.

Chapman, H.D. (2007). Rotation programmes for coccidiosis control. *Journal of International Poultry Production,* 15: 7 - 9.

Conway, D.P. and McKenzie, M.E. (2007). Poultry Coccidiosis*:* Diagnostic and Testing Procedures. 3rd Ed. Blackwell Publishing. Ames, IA, USA. Pp. 1 - 164.

DeGussein, M. (2007). 16th EU symposium on poultry nutrition. In: Coccidiosis in poultry; Review on diagnosis, control, prevention and interaction with overall gut health. Pp. 253 - 259.

Elbahy, N.M., Mokhbattly, A.M. and Verginia, M.E. (2006). Comparative clinicopathological studies on garlic ethanolic extract and halofuginone compound against coccidiosis in chicken. *Journal of Veterinary Medicine*, 1(1): 1 - 15.

Fakae, B.B. (1987). Veterinary Parasitology. Scotland: Bath Press, Avon. Pp. 217 - 221.

George, J.W. (2001). The usefulness and limitations of hand-held refractometers in veterinary medicine: A historical and technical review. *Journal of Veterinary Clinical Pathology,* 30(4): 201 - 210.

Kachmar, J.F. (1970). Determination of blood haemoglobin by the cyanomethaemoglobin procedure. In: Tietz NW Ed. Fundamentals of Clinical Chemistry, W.B. Sanders Company, Philadelphia. Pp. 268 - 269.

Lawrie, A.S., McDonald, S.J., Purdy, G., Mackie, I.J. and Machin, S.J. (1998). Prothrombin time derived fibrinogen determination on Sysmex CA-6000. *Journal of Clinical Pathology*, 51(6): 462 – 466.

Long, P.L. and Joyner, L.P. (1984). Problems in the identification of species of *Eimeria*. *Journal of Protozoology*, 31: 535 – 541.

Long, P.L., Millard B.J., Joyner, L.P., Norton, C.C. (1976). A guide to laboratory techniques used in the study and diagnosis of avian coccidiosis. Folia Veterinary Latina, 6: 200 – 217.

McDougald, L.R., Fitz-Coy, S.H. (2008). Coccidiosis. In: *Diseases of Poultry*, 12th Ed. Edited: Saif, Y.M., Fadly, A.M., Glisson, J.R., McDougald, L.R., Nolan, L.K., Swayne, D.E. Ames, IA: Blackwell Publishing Professional. Pp. 1068 – 1085.

Morris, G.M. and Gasser, R.B. (2006). Biotechnological advances in the diagnosis of avian coccidiosis and the analysis of genetic variation in *Eimeria*. *Journal of Biotechnological Advances*, 24: 590 – 603.

Ngongeh, L.A., Onyeabor, A., Nzenwata, E. and Samson, G.K. (2017). Comparative Response of the Nigerian Indigenous and Broiler Chickens to a Field Caecal Isolate of *Eimeria* Oocysts. *Hindawi Journal of Pathogens*, 2017: 1 – 9.

Okonkwo, C.J., Ukonu C. E., Uwalaka E. C. and Okwara, N. (2019). An Evaluation of the Anticoccidial Potency of Some Commonly Used Anticoccidial Drugs in Broiler Industry in Abia State. *Journal of Global Veterinaria*, 21(2): 58 - 64.

Petrie, A. and Watson, P. (1999). Statistics for Veterinary and Animal Science. 1st Ed. The Black Well Science Ltd, United Kingdom. Pp. 90 – 115.

Preston – Mafham, R.A. and Sykes A.H. (1967). Changes in permeability of the mucosa during intestinal coccidiosis infections in the fowl. *Journal of Experientia*, 23(11): 972 - 973.

Schildt, C.W. and Herrick, C.A. (1955). The effect of Caecal coccidiosis on the motility of the digestive tract of the domestic fowl. *Journal of Parasitology*, 41: 18 – 23.

Shivaramaiah, C, John, R. B., Hernandez-Velasco, G. and Hargis, T. (2014) Coccidiosis: recent advancements in the immunobiology of *Eimeria* species, preventive measures, and the importance of vaccination as a control tool against these Apicomplexan parasites. Veterinary Medicine Research and Reports. Pp. 15 - 35.

Soulsby, E.J.L. (1982). Helminths Arthropods and

Protozoa of Domesticated Animals. 7th Ed. Bailliere Tindall publishers, London, UK, Pp. 573 - 574.

Stephens, J.F. (1965). Some Physiological Effects of Coccidiosis caused by *Eimeria necatrix* in the chicken. *Journal of Parastology*, 51(3): 331 – 335.

Wakelin, D., Rose, M.E., Hesketh, P., Else, K.J. and Grencis, R.K. (1993). Immunity to Coccidiosis: genetic influences on Lymphocytes and Cytokine responses to infection with *Eimeria vermiformis* in inbred mice. *Journal of Parasite Immunology*, 15: 11 – 19.

Wakenell, P.S. (2010). Hematology of chickens and turkeys. In: Veterinary haematology; Weiss, D.J., Wardrop, K.J.: editors. 6th Ed. John Willey and Sons. Ames. Iowa, USA. Pp. 957–967.

Williamson, R.A., Misson, B.H. and Davison, T.F. (1985). The effect of exposure to 40° on heat production and the serum concentrations of triiodothyronine, thyroxine, and corticosterone in immature domestic fowl. Houghton Poultry Research Station, Houghton, Huntingdon, Cambridgeshire PE17 2DA, England. 60(2): 178 – 186.

Table 1: Mean feed intake of broiler chickens

Days PI	Groups		
	A	B	C
7	518.9±7.8^{a}*	390.5±3.6^{b}	419.4±19.7^{cb}
14	596.3±11.8^{a}	511.9±6.9^{b}	521.3±11.8^{cb}
21	596.3±8.5^{a}	759.1±7.3^{b}	690.8±41.5^{cb}
28	695.0±65.4^{a}	841.1±16.9^{b}	821.7±30.2^{cb}
35	966.6±17.9	978.0±21.0	998.5±2.5^{NS}
42	959.7±73.3	991.4±53.2	1101.7±14.6
AT	4425.5±160.7	4472.1±70.6	4483.7±55.8

Different superscript letters indicate significant differences (p<0.05) in rows. NS: not significant. *: mean ± SEM. AT = Average total feed intake per broiler

Table 2: Mean weight of broiler chickens

Days PI	Groups		
	A	B	C
0	340.0±9.6*	338.67±5.7	336.44±4.5
7	722.0±11.0^{a}	433.0±5.0^{b}	502.3±7.9^{c}
14	966.7±16.7^{a}	727.7±5.3^{b}	730.7±9.9^{cd}
21	1492.0±12.7^{a}	1082.0±20.1^{b}	1127.7±22.3^{cb}
28	2072.3±50.7^{a}	1547.0±84.5^{b}	1666.7±50.7^{cd}
35	2391.7±50.7^{a}	1858.3±84.5^{b}	2108.3±50.7^{c}
42	3127.7±64.2^{a}	2366.7±88.2^{b}	2625.0±62.9^{c}

Different superscript letters indicate significant differences (p<0.05) in rows. NS: not significant. *: mean ± SEM

Table 3: Activity scores of broiler chickens.

Days PI	Groups		
	A	B	C
0	5.0±0.0*	5.0±0.0	5.0±0.0
4	5.0±0.0^{a}	3.7±0.3^{b}	3.7±0.3^{cb}
5	5.0±0.0^{a}	2.7±0.3^{b}	2.3±0.3^{cb}
6	5.0±0.0^{a}	1.0±0.0^{b}	1.0±0.0^{cb}
7	5.0±0.0^{a}	1.0±0.0^{b}	2.0±0.0^{c}
8	5.0±0.0^{a}	1.3±0.3^{b}	1.3±0.3^{cb}
9	5.0±0.0^{a}	1.6±0.0^{b}	1.7±0.3^{cb}
10	5.0±0.0^{a}	2.0±0.0^{b}	3.0±0.0^{c}
11	5.0±0.0^{a}	2.7±0.3^{b}	3.3±0.3^{cb}
12	5.0±0.0^{a}	3.0±0.0^{b}	3.7±0.3^{c}
13	5.0±0.0^{a}	3.0±0.0^{b}	4.0±0.0^{c}
14	5.0±0.0^{a}	4.0±0.0^{b}	4.0±0.0^{ab}
15	5.0±0.0^{a}	4.0±0.0^{b}	4.3±0.3^{cb}
16	5.0±0.0^{a}	4.0±0.0^{b}	4.7±0.3^{ac}
17	5.0±0.0^{NS}	4.3±0.3^{NS}	5.0±0.0^{NS}

Different superscript letters indicate significant differences (p<0.05) in rows. Superscript ^{NS}= not significant. Day= day of life. *= mean ± SEM

Table 4: Faecal scores of broiler chickens

Days PI	Groups		
	A	B	C
0	5.0±0.0*	5.0±0.0	5.0±0.0
4	5.0±0.0^{a}	1.7±0.3^{b}	1.3±0.3^{c}
5	5.0±0.0^{a}	1.0±0.0^{b}	1.0±0.0^{cb}
6	5.0±0.0^{a}	1.0±0.0^{b}	3.0±0.0^{cb}
7	5.0±0.0^{a}	1.7±0.0^{b}	2.0±0.0^{cb}
8	5.0±0.0^{a}	1.0±0.0^{b}	3.3±0.3^{c}
9	5.0±0.0^{a}	2.0±0.0^{b}	2.0±0.3^{cb}
10	5.0±0.0^{a}	3.0±0.0^{b}	3.3±0.0^{cb}
11	5.0±0.0^{a}	3.0±0.0^{b}	3.0±0.0^{cb}
12	5.0±0.0^{a}	4.0±0.0^{b}	4.0±0.3^{cb}
13	5.0±0.0^{a}	4.0±0.0^{b}	4.0±0.0^{cb}
14	5.0±0.0^{a}	4.0±0.0^{b}	5.0±0.0^{ab}
15	5.0±0.0	5.0±0.0	5.0±0.0
16	5.0±0.0	5.0±0.0	5.0±0.0
17	5.0±0.0	5.0±0.0	5.0±0.0

Different superscript letters indicate significant differences (p<0.05) in rows. *= mean ± SEM. 1=very pronounced brown faeces, 2= pronounced brown faeces, 3 = brown faeces, 4 = slightly brown faeces and 5 =absence of brown faeces

Table 5: Plasma protein and fibrinogen of broiler chickens

Days PI	Group	P/Protein (g/dl)	Fibrinogen (mg/dl)
0	A	5.0±0.4*	476.7±62.3*
	B	4.9±0.5	430.0±25.2
	C	4.7±0.1	502.7±55.4
3	A	4.5±0.2^{a}	503.7±60.7^{a}
	B	3.7±0.1^{b}	710.0±20.8^{b}
	C	4.7±0.2^{ac}	710.0±45.8^{cb}
7	A	3.4±0.3^{a}	533.3±35.3
	B	3.4±0.0^{ab}	616.7±60.1
	C	2.0±0.3^{c}	646.7±72.2
10	A	3.0±0.2^{a}	450.0±51.3
	B	3.6±0.1^{b}	486.7±13.3
	C	3.5±0.6^{bc}	556.7±64.4
14	A	4.8±0.6^{a}	330.0±50.1^{a}
	B	4.1±0.7^{b}	350.0±50.0^{ab}
	C	3.8±0.2^{bc}	483.3±72.7^{c}
17	A	3.7±0.2	283.3±40.1^{a}
	B	3.7±0.1	366.7±33.3^{ab}
	C	4.3±0.3	460.0±83.3^{c}
21	A	3.8±0.2	360.0±70.2
	B	3.4±0.2	476.7±72.2
	C	4.0±0.2	340.0±30.6
24	A	3.5±0.4	575.0±52.0
	B	3.2±0.1	403.3±8.2
	C	3.5±0.2	466.7±88.2
28	A	3.6±0.3	183.3±40.1
	B	3.5±0.2	250.0±76.4
	C	3.5±0.3	250.0±76.4
35	A	4.1±0.1	266.7±120.2
	B	4.1±0.3	216.7±44.2
	C	3.4±0.2	216.7±16.7
42	A	3.8±0.0	283.3±101.4
	B	3.5±0.3	233.3±88.2
	C	3.7±0.2	366.7±66.7

Different superscript letters indicate significant differences (p<0.05) in columns. PI.: post -infection. *: mean ± SEM.

Table 6: The haematological profile (mean ± SEM) of the broilers

Days PI	Group	RBC (x10^{6}µl)	HB (g/dl)	PCV (%)	WBC (x10^{3}µl)
0	A	2.8±0.1	11.5±1.2	24.7±1.3	48.9±5.9
	B	2.8±0.2	12.3±0.1	25.0±0.0	30.9±1.8^{b}
	C	2.8±0.1	10.3±1.1	24.0±0.6	36.5±1.1^{ac}
3	A	3.1±0.2	12.4±1.2	27.3±1.8	34.5±3.5
	B	3.0±0.1	11.1±1.2	25.3±0.9	29.3±1.9
	C	3.0±0.2	12.4±0.7	25.3±0.3	35.2±1.1
7	A	3.7±0.4^{a}	11.5±1.2	34.0±2.1^{a}	33.6±2.8
	B	3.6±0.3^{ab}	12.3±0.1	22.3±5.4^{ab}	34.1±2.3
	C	2.3±0.2^{c}	10.3±1.1	19.0±2.9^{cb}	26.2±4.1
10	A	3.3±0.1^{a}	13.7±0.1	31.7±1.9^{a}	31.1±1.0^{a}
	B	2.4±0.1^{b}	8.3±0.1	21.0±0.6^{b}	25.1±0.9^{b}
	C	2.6±0.1^{cb}	8.5±0.2	22.0±0.6^{cb}	32.7±1.6^{ac}
14	A	3.4±0.1	12.5±0.1	29.7±0.3	36.0±0.8^{abc}
	B	3.6±0.2	13.1±0.3	31.3±0.9	32.3±1.8^{b}
	C	3.6±0.3	13.3±0.4	31.7±1.8	38.5±1.3^{ac}
17	A	2.7±0.1^{a}	9.9±0.4^{a}	37.7±3.4^{a}	37.0±5.9
	B	3.4±0.2^{b}	12.7±0.9^{b}	30.0±1.7^{b}	34.6±1.8
	C	3.2±0.2^{cb}	11.7±0.9^{cb}	31.3±2.3^{cb}	35.2±1.1
21	A	2.8±0.2	11.7±0.6	36.7±1.8^{a}	34.1±0.7^{a}
	B	3.1±0.1	12.3±0.4	28.3±0.9^{b}	37.3±0.8^{b}
	C	3.2±0.3	12.4±0.7	28.7±1.5^{cb}	35.5±0.7^{abc}
24	A	2.8±0.1	11.4±0.3	32.7±0.3^{a}	35.5±0.4^{abc}
	B	3.0±0.1	12.0±0.1	28.7±1.3^{b}	33.1±0.9^{b}
	C	2.8±0.2	11.27±0.3	27.7±0.3^{cb}	37.1±1.0^{ac}
28	A	3.2±0.1	12.4±0.2	31.3±0.9	34.9±1.7^{abc}
	B	3.5±0.3	13.2±0.4	32.0±1.7	38.5±1.1^{b}
	C	3.1±0.1	12.4±0.1	29.7±0.7	33.1±1.1^{ac}
35	A	3.4±0.2	12.0±0.3	30.7±0.9	40.4±1.2^{a}
	B	3.2±0.3	11.7±0.5	27.3±2.0	36.5±0.4^{bc}
	C	2.7±0.3	11.5±0.2	27.7±1.2	35.5±0.3^{cb}
42	A	3.6±0.2^{a}	12.5±0.3^{a}	32.7±0.3	31.4±0.6
	B	2.9±0.3^{b}	11.6±0.1^{b}	28.7±1.3	32.5±1.3
	C	2.9±0.3^{cb}	11.7±0.2^{cb}	27.2±0.9	35.2±1.7

Different superscript letters indicate significant differences (p<0.05) in columns. RBC red blood cell. Hb haemoglobin, WBC white blood cell, PCV= packed cell volume, PI= post-infection

Table 7: *Eimeria* oocyst counts of experimental broiler chickens

Days PI	Groups		
	A	**B**	**C****
0	0	0	0
5	0^{a}	384.0±5.0^{b*}	347.3±69.3^{cb}
6	0^{a}	1233.3±202.8^{b}	1592.5±339.5^{cb}
7	0^{a}	1366.7±202.8^{b}	1592.0±339.5^{cb}
8	0^{a}	2366.7±202.8^{b}	1900.0±237.0^{cb}
13	0^{a}	2133.3±202.8^{b}	1453.3±50.0^{c}
18	0^{a}	2869.3±202.8^{b}	1366.7±260.3^{c}
19	0^{a}	1696.3±207.4^{b}	618.0±463.5^{ca}

Different superscript letters indicate significant differences (p<0.05) in rows. *= mean ± SEM. ** = Birds were treated on day 6 PI

Plate 1: PM lesion of group B with the caeca showing balloon and haemorrhage (B-D) and healing stage (E-F), bloody faeces (A), bloody caecal core (D).

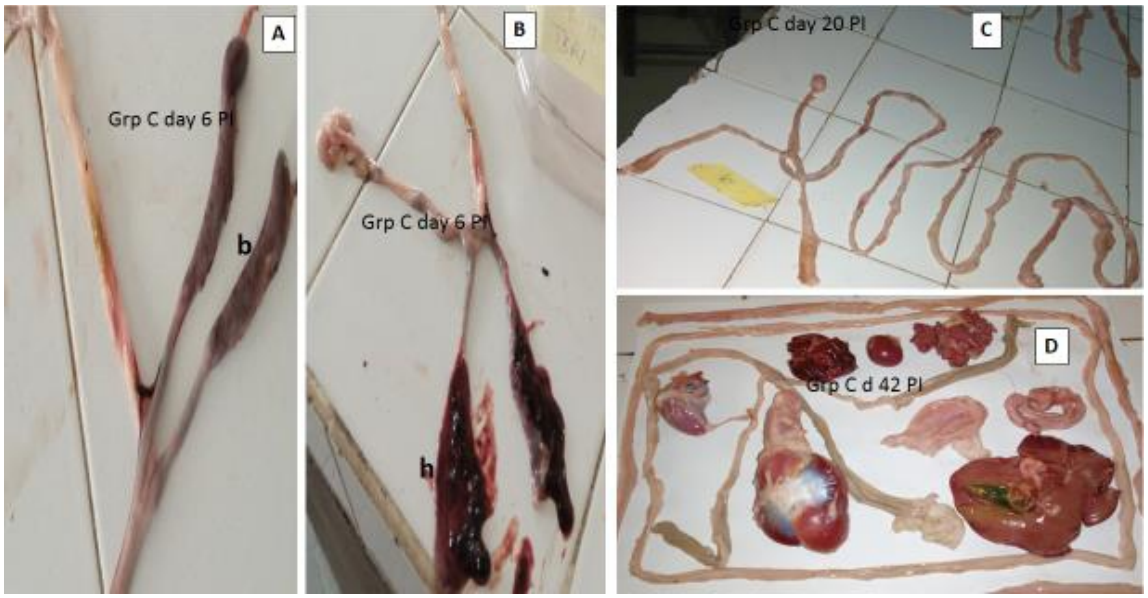

Plate 2: Group C birds with PM lesion in the caeca showing balloon (b) in A and haemorrhagic core (h) in B. Healing stages in C at day 20 PI and D at day 42 PI.

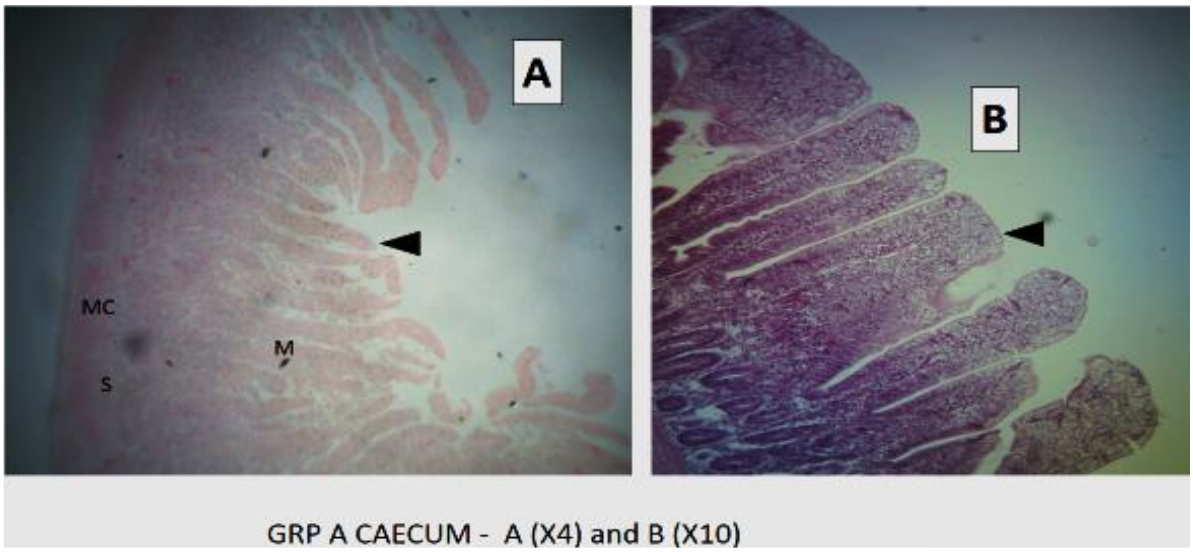

Plate 3: Caecal histology of an uninfected control broiler (Group A). H&E SHOWING INTACT EPITHELIA (arrow head) in A and B. MC= muscularis, S=submucosa and M=mucosa.

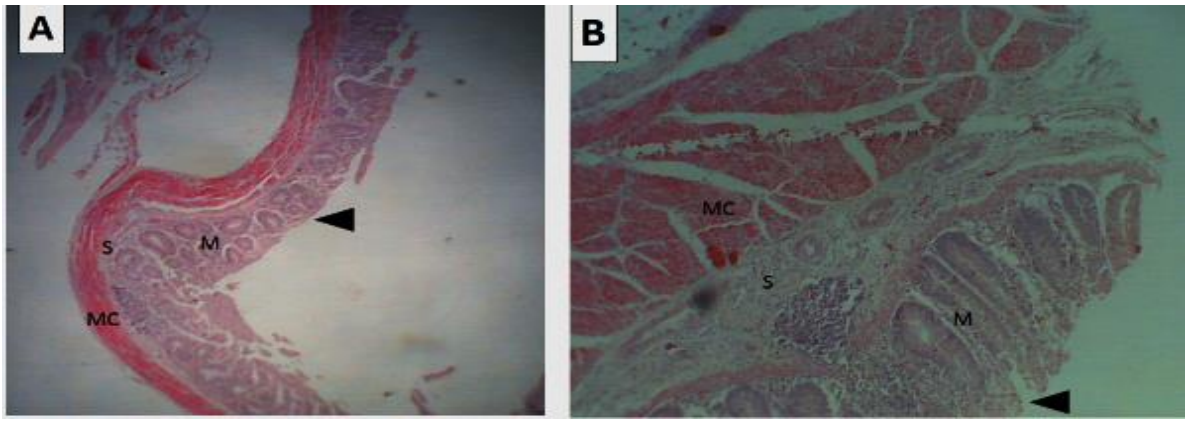

Plate 4: Grp A Ceaca A X4 mag, B X10 showing intact mucosa (arrow head), mucosa (M), submucosa (S) and muscularis layer (MC)

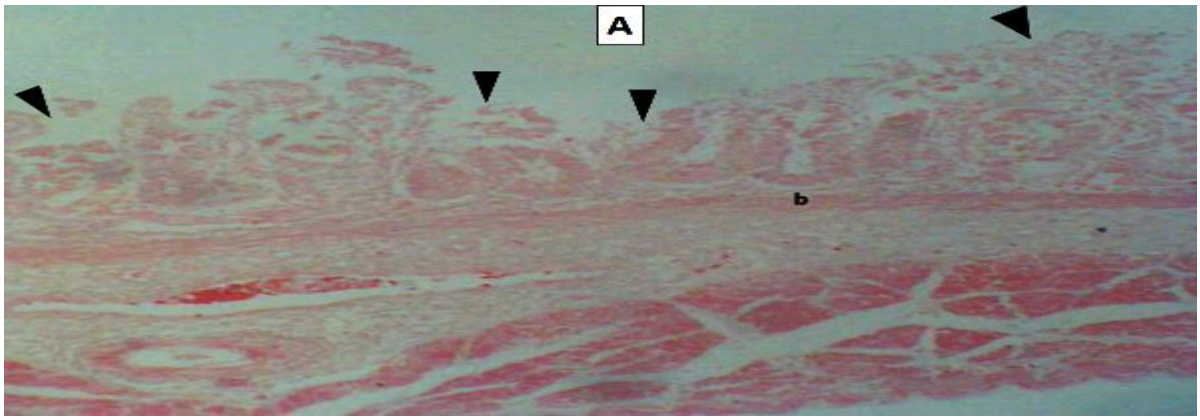

Plate 5: Grp B Ceacum – X10 showing ulceration (arrow head) and disorganization of the mucosa

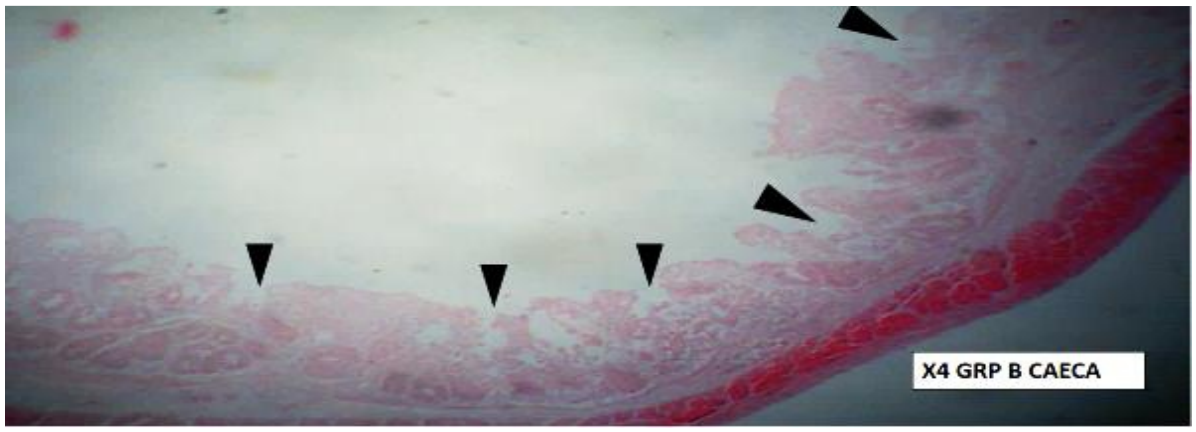

Plate 6: Grp B Ceacum – X4 showing ulceration (arrowhead) and disruption of the mucosa

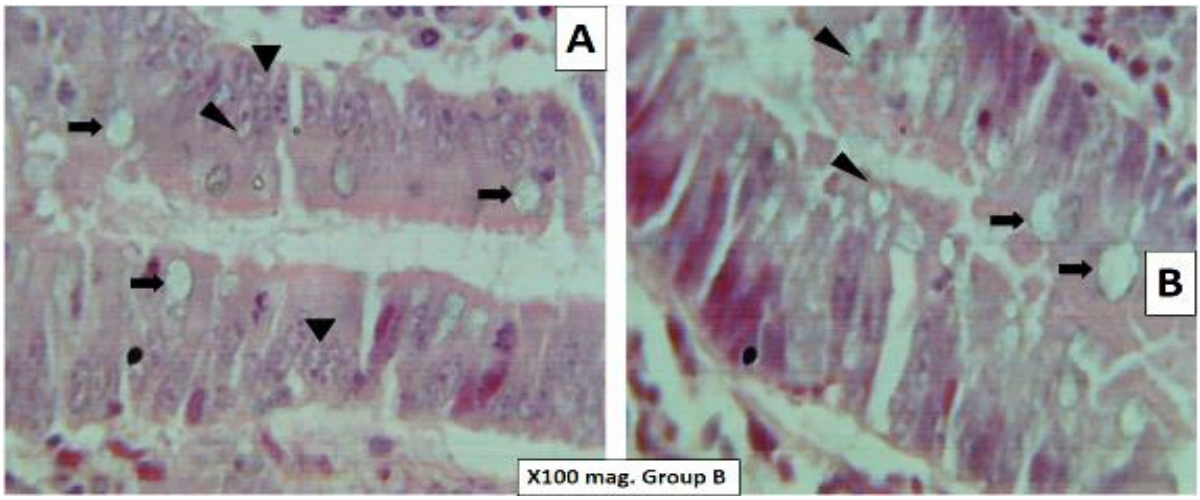

Plate 7: Grp B Ceacum – X100 showing oocyst (arrow) and schizonts (arrow head) of the mucosa

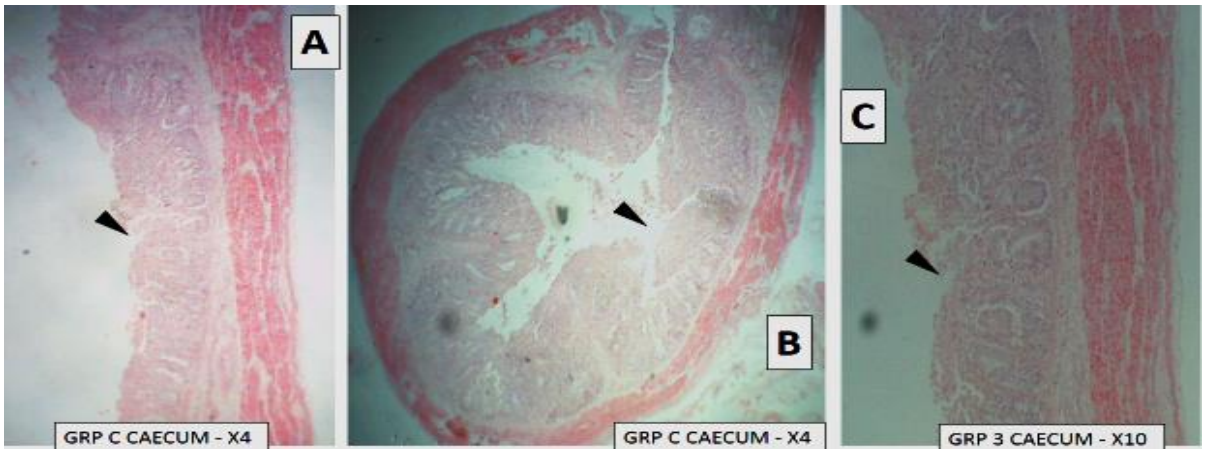

Plate 8: Grp C Ceacum – X100 showing erosion (arrowhead) of the mucosa in A, B and C

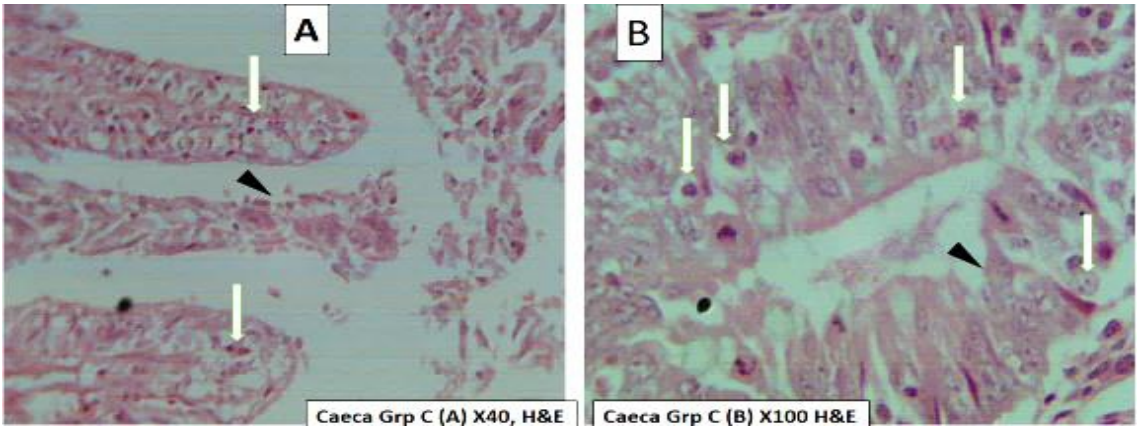

Plate 9: Grp C Ceacum showing developmental stages (arrow) of Eimeria in the mucosa in A and B and sloughing of epithelia (arrowhead).

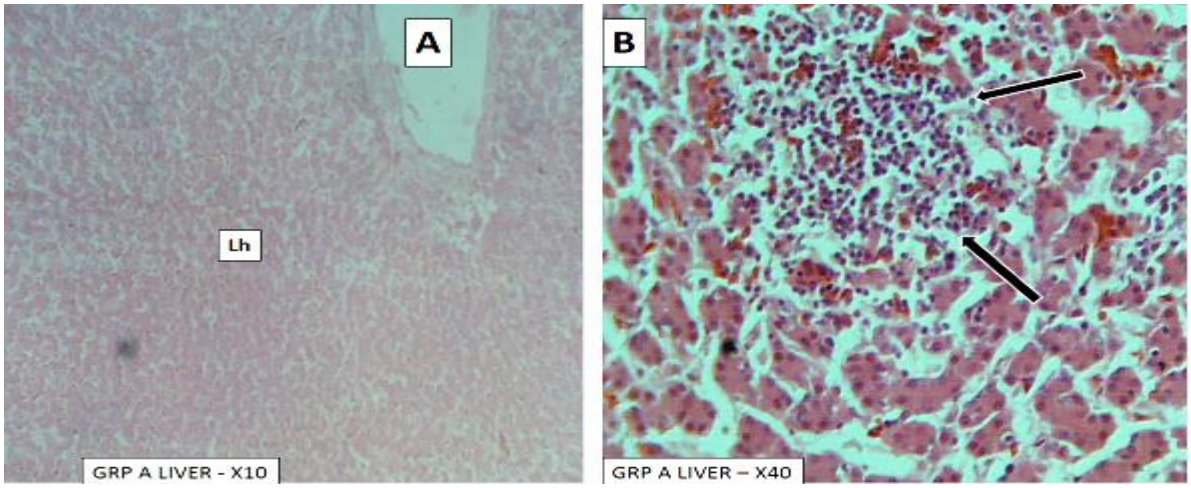

Plate 10: Liver hepatocyte (Lh) arranged in cords in A and lymphoid nodule (arrow) in B

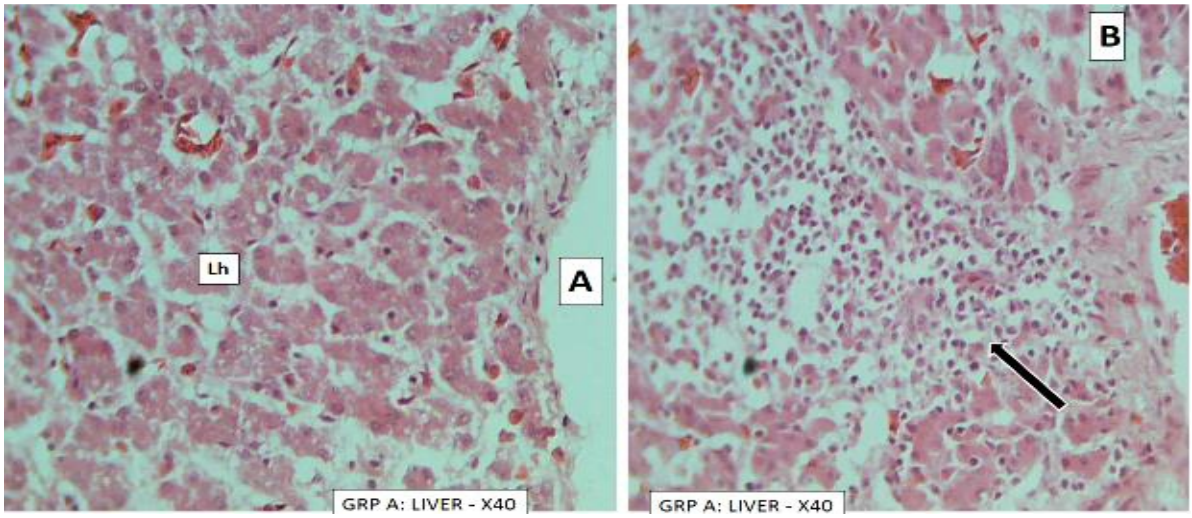

Plate 11: Liver hepatocyte (Lh) arranged in cords in A and lymphoid nodule (arrow) in B

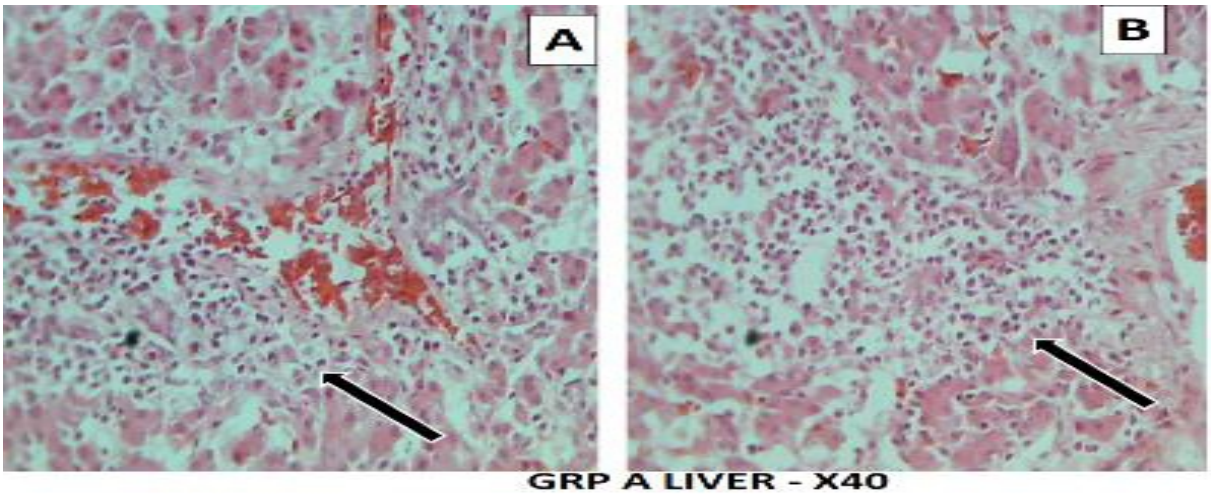

Plate 12: Liver showing lymphoid nodule (arrow) in A and B

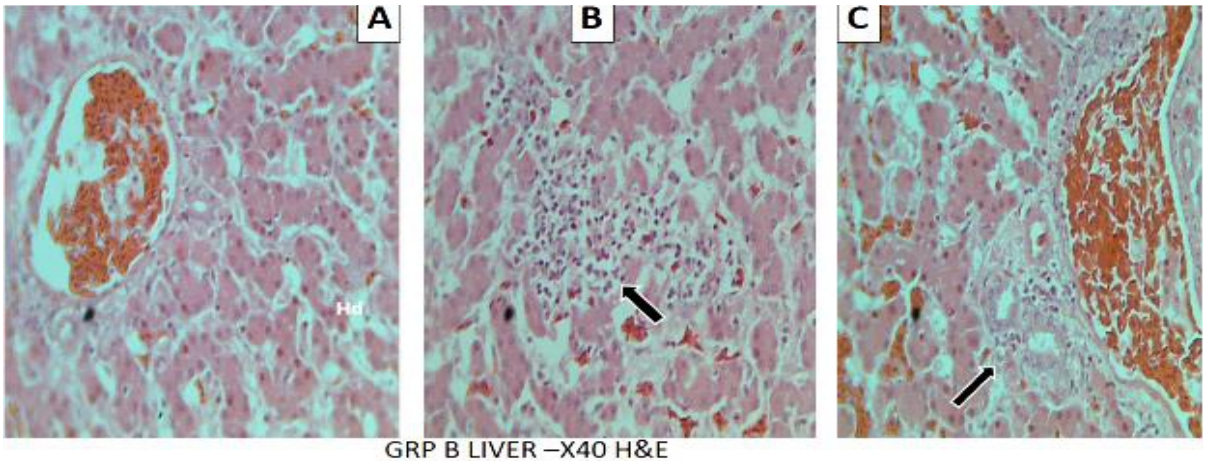

Plate 13: Liver showing depletion of lymphoid nodule (arrow) in B and C. A shows disorganization (Hd) of hepatic cords.

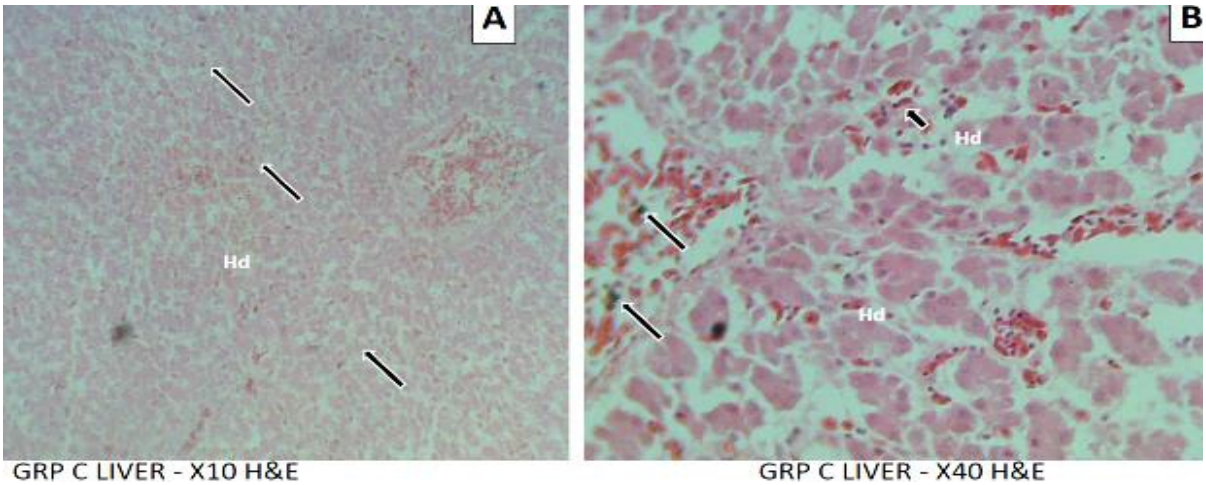

Plate 14: Liver showing disorganization (Hd) of hepatic cords and marked hemosiderosis (arrow) in A and B.