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Sequential pathology of fowl adenovirus serotype 11 in experimentally infected chicken

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ABSTRACT

The present study was undertaken to find out the sequential pathology of fowl adenovirus in experimentally infected chicken. To assess the gross and histopathology, experimental infection of ninety-eight numbers of one-day-old chicks were inoculated with fowl adenovirus (FAdV) serotype 11 field isolate at the rate of 0.5 ml of $10^{6.5}$/$TCID_{50}$ per ml both oral and intramuscular (I/M) route except control group, followed by PCR confirmation and sequencing. Six number of birds from each treatment groups and two number of birds from control groups were sacrificed with 3, 5, 7, 10, 14, 17 and 21st days post infection (dpi) and detailed post mortem examination was conducted. Tissue samples viz., bursa of Fabricius, caecal tonsil, kidneys, liver, spleen and thymus were collected in 10 per cent formalin in sterile container for histopathological examination. Gross pathological lesions were observed in liver from 3rd dpi to 21st dpi in both the groups. Enlarged and congested kidneys were seen at 5th dpi and 7th dpi in I/M and oral group respectively. The intensity of lesions in liver, heart and kidneys were slightly higher in I/M group than the oral group. Histopathological examination of infected tissues revealed mild to severe changes were observed in all tissues except in thymus. The presence of basophilic intranuclear inclusion bodies were found in I/M group from 7th dpi whereas 21st dpi in Oral groups.

Keywords: Experimental infection, Fowl adenovirus, Histopathological examination, Sequential gross

Fowl adenoviruses (FAdVs) infection causes number of disease conditions in chicken, including inclusion body hepatitis (IBH), hydropericardium syndrome (HPS), hepatitis – hydropericardium syndrome (HHS), gizzard erosions (GE), proventriculitis (PV), tenosynovitis and respiratory infections in poultry (McFerran and Smyth 2000). Fowl adenoviruses are classified under the family *Adenoviridae,* genus *Aviadenovirus* and it includes eight species, five of which are the FAdV species consisting of *Fowl Aviadenovirus A* (FAdV-1); *Fowl aviadenovirus B* (FAdV-5); *Fowl aviadenovirus C* (FAdV-4 and 10) *Fowl aviadenovirus D* (FAdV-2, 3,9 and 11) and *Fowl aviadenovirus E* (FAdV-6, 7, 8a and 8b) (Harrach *et al.* 2012). Recently, IBH and HHS have been widely distributed in broiler flocks in several countries and there has been a trend toward more epidemic breakouts rather than sporadic epidemics. Furthermore, the significant mortality and growth retardation associated with IBH and HHS have resulted in enormous economic losses (Marek *et al*. 2014, Schachner *et al*. 2018). In India, IBH is usually associated with FAdV-2 and 11 (species D) (Chitradevi *et al*. 2021). IBH has been documented in chickens as young as one and four days old and typically affects poultry between the ages of 3–5 weeks (Pilkington *et al*. 1997, Chitradevi *et al*. 2021). The affected birds showed varying degrees of mortality, lethargy, ruffled feathers and diarrhoea (Singh *et al*. 1996, Gaba *et al*. 2010, Thakor *et al*. 2012). Macroscopically, affected birds usually show pale yellow, friable and enlarged livers with varying degrees of petechial and/or ecchymotic haemorrhages and enlarged and congested kidneys (Kumar *et al*. 2013, Chen *et al*. 2019, Zhao *et al*. 2015). Histologically, liver exhibited necrosis of hepatocytes, vacuolar degeneration and infiltration of mono nuclear cells and presence of eosinophilic or basophilic intranuclear inclusion bodies (Balachandran *et al*. 1993, Kumar *et al*. 2013). Diagnosis of IBH can be carried out through conventional methods by observation of gross and histopathological changes, virus isolation and molecular techniques like hexon gene-specific polymerase chain reaction and sequencing have been used for rapid detection FAdVs (Raue and Hess 1998, Meulemans *et al*. 2001, Thakor *et al*. 2012, Pereira *et al*. 2014, Norfitriah *et al*. 2018, Abghour *et al*. 2021). Hence, the present study was undertaken to find out the sequential pathology of field isolated and characterized fowl adenovirus serotype 11 in experimentally infected chicken and this study will helps to find out the pathogenesis pattern of FAdV serotype 11.

MATERIALS AND METHODS

Virus Strain: The FAdV strain used in this study was

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isolated from liver samples of broiler chicken suffering with IBH in Tamil Nadu, India and it was molecularly identified by PCR against group specific hexon gene by using Hexon A and Hexon B primers for 890 bp hexon gene (Meulemans *et al*. 2001) as belonging to FAdV-D serotype (GenBank accession number MK816403.1).Virus isolatewas also checked against concurrent infection for Marek's disease virus (MDV), Avian leucosis virus (ALV), Reticular endothelial virus (REV) and Chicken infectious anaemia virus (CAV). The field isolate was scaled up using primary chicken embryo liver cells and $TCID_{50}$ assay was calculated as per the method of Reed and Muench (1938).

Experimental study: All animal procedures were performed in accordance with local ethical regulations and the approval of in - house Ethical Committee. During the experimental period of three weeks, the chicks were housed in an in-house animal facility unit and effort was taken to minimize the stress in chicks.

To study the pathology of chickens infected with FAdV 11, 98 one day old broiler chicks were divided into three groups viz. Group I (42 chicks), Group II (42 chicks) and Group III (14 chicks - uninoculated control). Group I and group II chickens were inoculated with 0.5 mL of chicken embryo liver cell culture fluid containing $10^{6.5}/TCID_{50}$ per ml FAdV 11 isolated by oral and intramuscular route, respectively. The birds were observed daily for mortality and clinical signs up to 21 days post infection (dpi). Six birds from each treatment groups and two birds in control group were sacrificed as per the required procedure on 3, 5, 7, 10, 14,17 and 21st dpi and gross lesions were recorded.

Gross and histopathological examination: During experimental study period, birds of each group were observed the development of abnormal clinical and behavioural signs up to 21st day of post infection. Detailed post mortem examination was conducted and gross pathological changes were noted carefully and the tissue samples viz., *bursa of Fabricius*, caecal tonsils, kidneys, liver, spleen and thymus were collected and stored in 10 per cent formalin for histopathological examination. The formalin fixed tissues were processed by paraffin wax embedding method for tissue sectioning. The sections were cut at 5-6 microns thickness with automatic section cutting machine (Rotary Microtome RM 2125, Leica, China) and were stained with haematoxylin and eosin (H&E) stain (Bancroft and Stevens 1996). The H & E stained slides were read under microscope and histopathological changes were recorded.

RESULTS AND DISCUSSION

In recent years, FAdV infections in chickens have increased and many FAdV strains related to IBH have been identified in many countries including in India (Trivedi *et al*. 2018, Shinde *et al.* 2020, Chitradevi *et al*. 2021, Shankar *et al*. 2022, Chavan *et al*. 2023). In the present study, FAdV-11 strain isolated from field outbreak in Tamil Nadu, India was used to find out the sequential pathology and tissue tropism following experimental infection of broiler chickens through oral and intramuscular route. Fowl adenovirus strain 11 used in this study was confirmed by PCR (Fig. 1). The phylogenetic analysis also revealed that the FAdV belongs to FAdV D serotype 11 (Gaba *et al*. 2010, Thakor *et al*. 2012, Pereira *et al.* 2014).

In experimental infection, no mortality could be observed from all three groups for entire study period. Mild clinical signs include dullness, depression and diarrhoea were noticed between 3rd to 5th dpi and not in control group. No other major clinical signs were observed. The result of this study is in accordance with the findings of Jadhao *et al*. (2003), Grgic *et al*. (2011) and Steer e*t al*. (2015) Though FAdV acted as primary pathogen for causing IBH, absence of concurrent infection, differences in environmental conditions between field and experimental studies along with other stress associated with farm condition, and difference in susceptibility of chicken breeds used in study might influence the outcome of the disease.

On necropsy examination, enlarged, pale, friable liver with haemorrahgic cyst was observed from 3rd to 21st dpi from both the treatment groups of birds and gross lesions in liver was consistent throughout the study (Fig. 2). Accumulation of 0.5 to 1 mL of straw yellow colour fluid in pericardial sac was found from 3rd dpi in I/M group whereas in oral group it was started from 7th dpi (Fig. 3). Enlarged and congested kidneys were seen at 5th dpi and 7th dpi in I/M and oral group respectively (Fig. 4). No gross lesions were observed in other organs such as bursa of Fabricius, caecal tonsil, spleen and thymus as well as control group birds. Based on the extent and severity of gross lesions, it was observed that prominent liver pathology was observed at 10th dpi after that gradual decline in intensity of lesion in liver and heart at 21st dpi in both the groups was observed. The intensity of lesions in liver, heart and kidneys were slightly higher in I/M group than the oral group. Similarly, Steer *et al*. (2015) observed no gross lesions in the liver of any birds from day 1 to 3 dpi and multiple gross liver lesions were seen in 9 out of 12 FAdV 11 inoculated birds examined between 4 to 7 dpi.

The gross lesions observed in this study is well supported by Alcigir and Vural (2013) who experimentally infected four weeks old healthy broiler chicks with 0.5 ml of $10^{7.3}$ log EID_{50}FAdV serotype 4 by oral and I/M

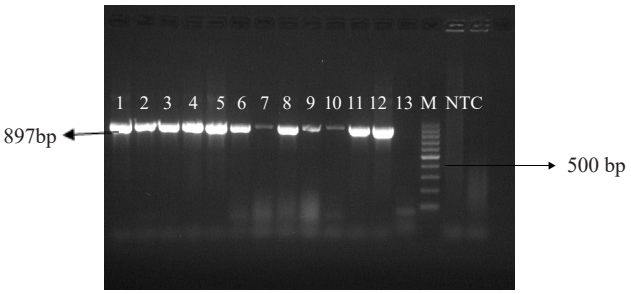
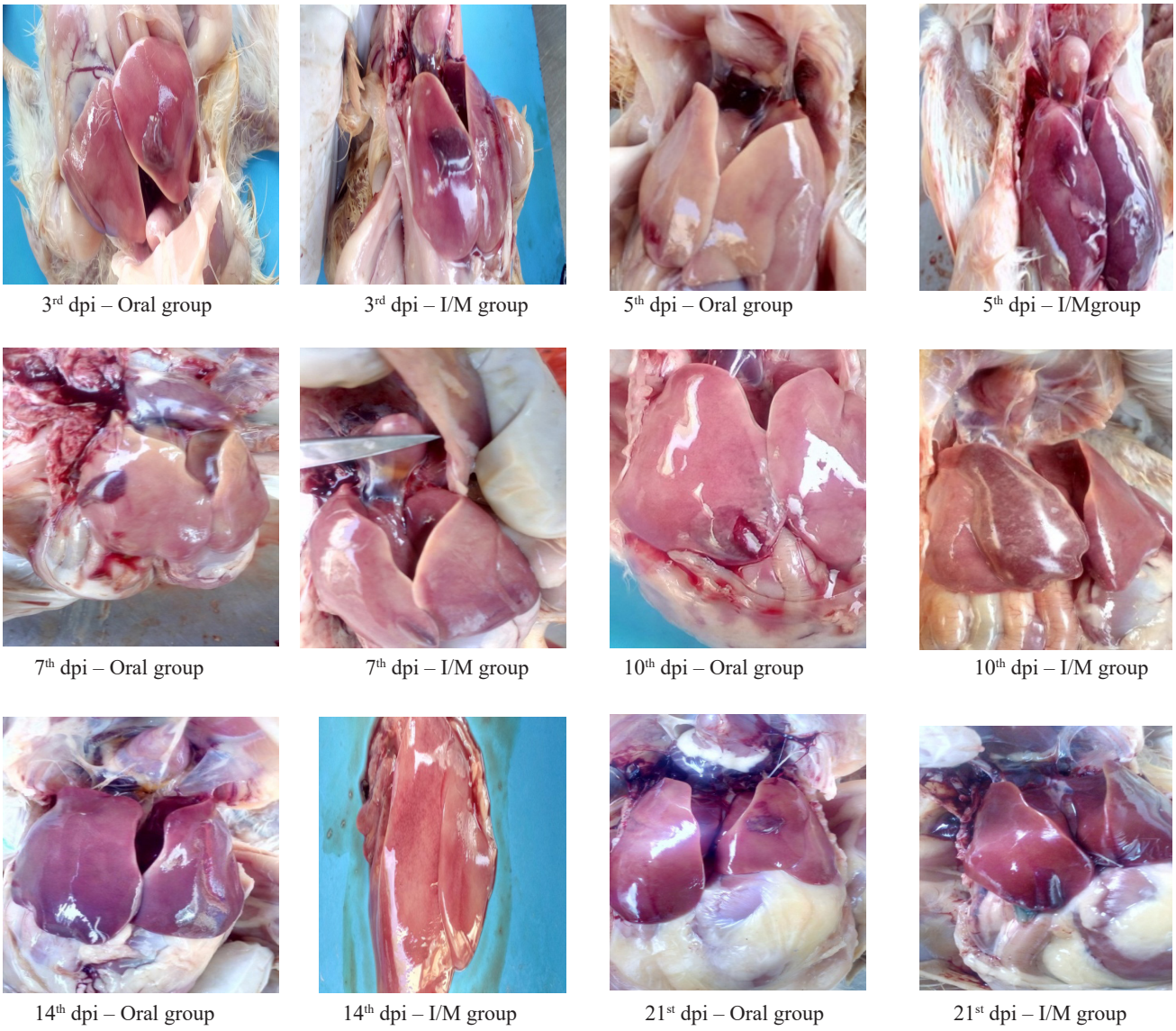


Fig. 1. Agarose gel electrophoresis showing 897 bp amplified PCR product of hexon gene of field FAdVs 1 to 13 field samples, M: Marker, NTC: No template control

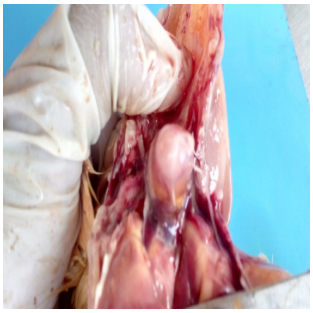


3rd dpi – Oral group | 3rd dpi – I/M group | 5th dpi – Oral group | 5th dpi – I/Mgroup

7th dpi – Oral group | 7th dpi – I/M group | 10th dpi – Oral group | 10th dpi – I/M group

14th dpi – Oral group | 14th dpi – I/M group | 21st dpi – Oral group | 21st dpi – I/M group

Fig. 2. Enlarged pale, friable liver with varying sizes haemorrahgic cyst in oral and I/M group during experimental study period



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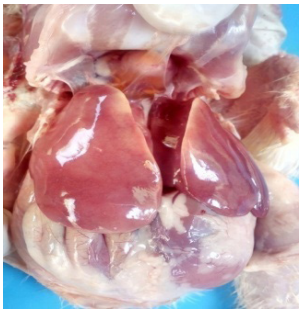
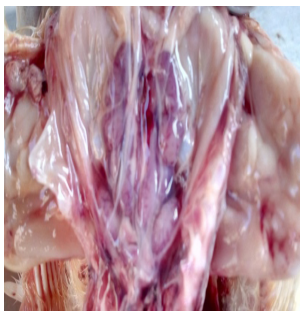
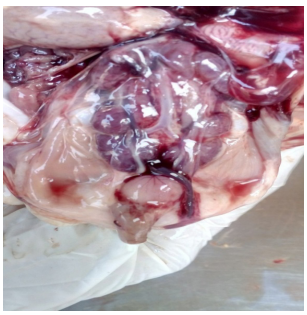


(b)

Fig. 3. (a) Hydropericardium at 3rd dpi in I/M group and (b) 7th dpi in oral group



(a)



(b)

Fig. 4. Enlarged and congested kidneys at (a) 5th dpi in I/M group and (b) 7th dpi in oral group

route and found no mortality in chicks but degenerative and inflammatory changes were found in heart and liver. They also observed the intensity of the organ damage was greater in intramuscularly infected birds than the orally infected birds. Similarly, Zhao *et al*. (2015) observed mild to severe hepatitis and hydropericardium when the chicks were infected with HBQ12 strain of FAdV serotype 11.

Histopathological examinations of liver, kidney and

lymphoid organs such as *bursa of Fabricius*, caecal tonsil, spleen and thymus were carried out in the experimentally infected chicken. Mild to severe lymphoid depletion, hyperplasia of plical epithelium, necrosis of cortical lymphoid tissues, fibrous tissue hyperplasia in inter follicular area were observed in *bursa of Fabricius* from 3rd to 21st dpi (Fig. 5). In caecal tonsil, depletion and lymphoid cell necrosis were observed throughout the study period (Fig. 6). There was mild lymphoid depletion, fibrous tissue proliferation in peri articular lymphoid sheath (PALs), proliferation of fibrous tissues in peri ellipsoidal lymphoid tissue (PELT) and haemorrhages were noticed in spleen on 10th and 14th dpi only (Fig. 7). The result of this study is well correlated with Steer *et al*. (2015), who observed lymphoid depletion in *bursa of Fabricius*, spleen and thymus when the birds were inoculated with FAdV 11. The lymphoid depletion observed in the lymphoid organs in the present study clearly showed that the FAdV could cause immunosuppression. These finding were well supported by Schonewille *et al*. (2008) who reported pathogenic FAdVs could suppress the humoral and cellular response of chickens.

In liver, severe congestion, moderate to severe vacuolar degeneration, mononuclear cells infiltration, hyperplasia of biliary epithelium, moderate to severe sinusoidal congestion, fatty changes varying from micro to macro vesicles, periductular infiltration of mononuclear cells were observed from 3rd to 21st dpi in both I/M and oral route

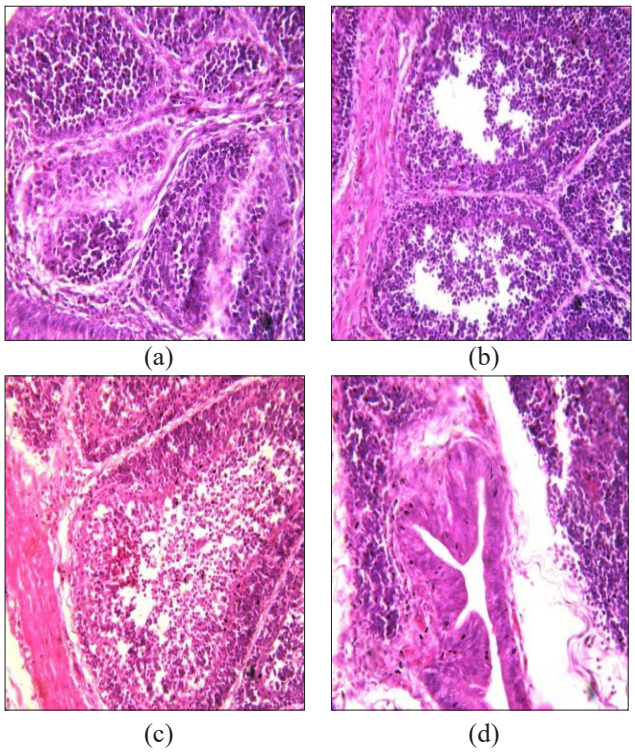


Fig. 5. *Bursa of Fabricius* (a) and (b)Mild to severe depletion of lymphoid cells, (c) indistinct demarcation of cortex and medulla and (d) hyperplasia of plical epithelium from 3rd to 21st dpi (H&E 100X)

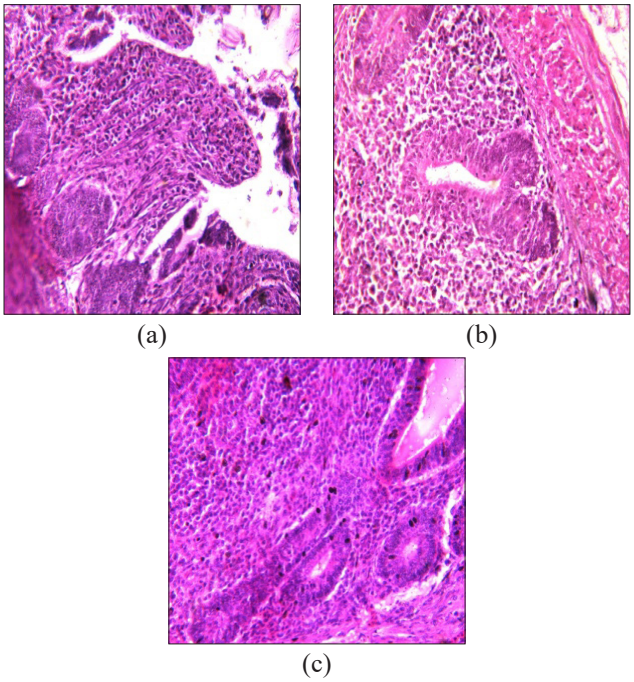


Fig. 6. Caecal tonsil: (a) Mild, (b) moderate and (c) severe hyperplasia of lymphoid cells from 3rd to 21st dpi (H&E 100X)

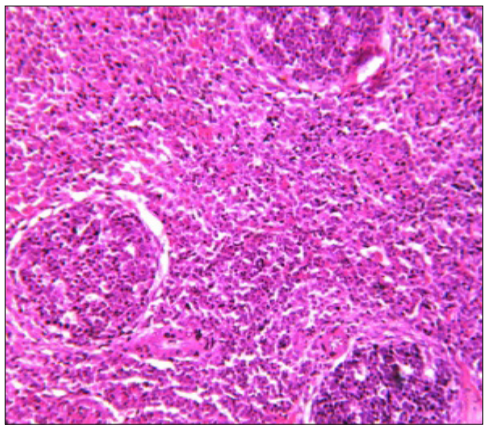
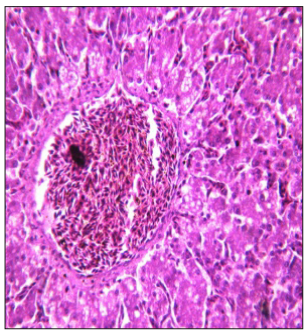


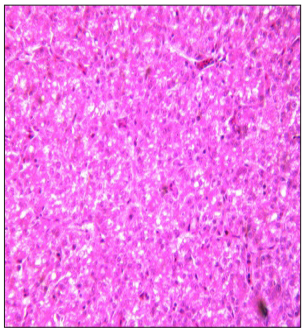
Fig. 7. Regenerative lymphoid nodule, moderate haemorrahge and congestion in Spleen (H&E 100X)

of infection. Initially basophilic intranuclear inclusion bodies (INIB) were first appreciated at 5th and 7th dpi in intramuscularly and orally infected birds respectively. After that, it was noticed up to 21st dpi in hepatocytes (Fig. 8).

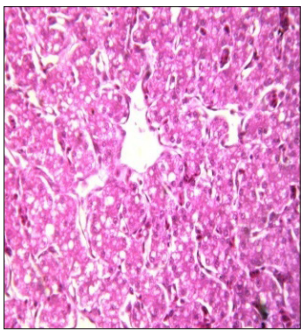
In both the groups, kidneys showed moderate degeneration, necrosis of tubular epithelium, severe degeneration, desquamation of tubular epithelium, presence of crystals within the tubules, perivascular fibrosis, interstitial haemorrhage and intertubular congestion were observed in entire study period (Fig. 9). The histopathologic lesions observed in liver and kidneys are similar to the observation of Zhao *et al*. (2015), who studied the pathogenicity of FAdV serotype 11 (HBQ12) and FAdV 4 (JSJ13). In the present study, the histopathological lesions observed in intramuscularly infected birds were severe



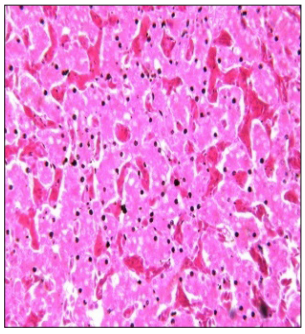
3rd dpi– Oral group– Severe congestion and vacuolar changes



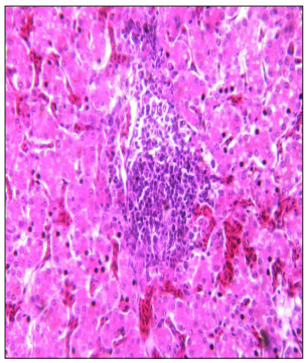
3rd dpi–I/M group-Diffuse vacuolar changes



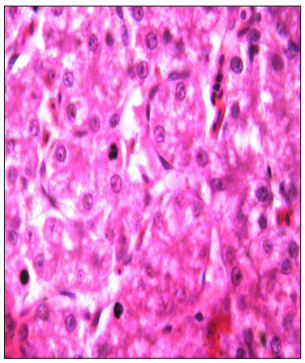
5th dpi– Oral group– Severe fatty changes (Macro and micro vesicles)



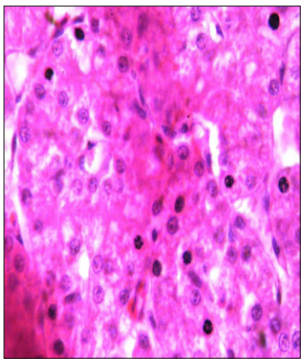
5th dpi– I/M group– Fatty changes and sinusoidal congestion



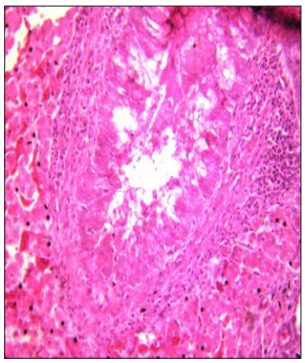
Liver-7th dpi – Oral group: – Focal infiltration, sinusoidal congestion and fatty changes



Liver - 10th dpi – I/M group – Basophilic intranuclear inclusion in hepatocytes

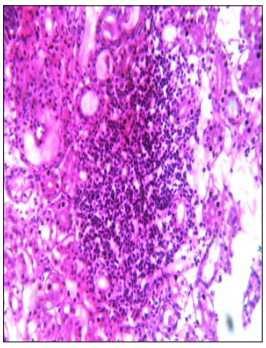


Liver – 14th dpi – Oral group – Basophilic intranuclear inclusion in hepatocytes

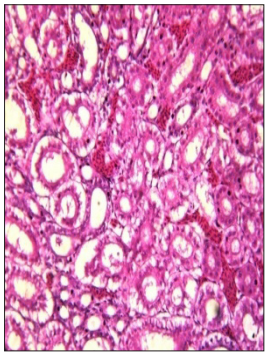


Liver – 14th dpi – I/M group Periductular fibrosis and mononuclear infiltration

Fig. 8. Histopathological lesions observed in liver during experimental study period from 3rd to 21st dpi (H&E 100X)



(a)



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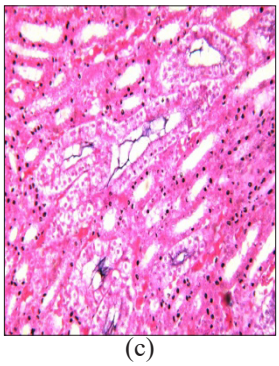

(c)

Fig. 9.(a) Massive aggregation of inflammatory cells and tubular necrosis, (b) moderate tubular degeneration and (c) inter-tubular haemorrhage and crystals in multiple tubules (H&E 100X)

when compared to orally infected birds and similar findings observed by Lim *et al*. (2011). Disruption of myocardial fibres and fibrous tissue replacement and infiltration of monocytes were noticed in heart. Many earlier researchers had similar observations during their experimental studies (Zhao *et al*. 2015, Suohu and Rajkumar 2021, Joshi *et al*. 2022).

In conclusion, though there were no recognizable gross lesions in *bursa of Fabricius*, caecal tonsil and spleen during entire study period, histological evidence revealed that the viral replication also occurred in lymphoid organs. Liver showed massive gross and histopathological lesions including the presence of basophilic intranuclear inclusions in hepatocytes. Significant lesions were also appreciated in kidneys. Gross pathological lesions were prominent in liver, kidneys and heart. Gross lesions in intramuscular group were started earlier. The and intensity of gross and histopathological lesions was also higher in intramuscularly infected birds when compared to orally infected chickens.

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