Indian J. Comp. Microbiol. Immunol. Infect. Dis.
Vol. 39 No. 1 (Jan-June), 2018: 20-22

Research Article

CONCURRENT INFECTIONS ASSOCIATED WITH FOWL ADENOVIRUS IN COMMERCIAL BROILER CHICKEN

S. Chitradevi*, K. Sukumar, P. Suresh, G.A. Balasubramaniam, D. Kannan and A. Raja
Department of Veterinary Microbiology, Veterinary College and Research Institute, Namakkal, Tamil Nadu

ABSTRACT

The objective of the present study was to find out the presence of concurrent viral infections associated with fowl adenovirus confirmed flock of commercial broiler birds. The liver samples were collected from 27 nos of FAdV confirmed commercial broiler farms. All the fowl adenovirus positive DNA was used to screen the Marek's disease virus (MDV), Avian leucosis virus (ALV), Reticular endothelial virus (REV) and Chicken infectious anaemia virus (CIAV). Out of 27 FAdV positive flocks, no flocks were positive for MDV and REV whereas 21 flocks (77%) were positive for ALV by PCR and 16 flocks (59%) were positive for CIAV by PCR. Though FAdV is a primary causative factor for IBH and HPS, the immunosuppression induced by ALV and CIAV plays a major role for aggravating the FAdV infection in broiler chicken. Further studies needed to elucidate the interaction of these viruses and their impact on the poultry health.

Keywords: Fowl adenovirus, Marek's disease virus, Avian leucosis virus, Chicken anaemia virus, PCR

Received: 20 March, 2018; Accepted: 28 May, 2018
DOI: 10.5958/0974-0147.2018.00005.3

INTRODUCTION

Fowl adenoviruses have a worldwide distribution and are reported to be frequently isolated from healthy chickens as well as affected birds (McFerran and Smyth, 2000). They are responsible for a wide range of clinical presentations, including hydropericardium syndrome (HPS), inclusion body hepatitis (IBH), respiratory tract disease, and gizzard erosion (McFerran and Adair, 2003). FAdV suspected commercial broiler birds show dullness, uneven growth, reduced feed intake and some flocks showed leg weakness (Gaba *et al*., 2010) and mortality ranging from 10-30 per cent (Dahiya *et al*., 2002) and which may reached up to 80 per cent (Kumar *et al*., 2003). FAdVs can be transmitted vertically through embryonated eggs and that the virus can be reactivated in chicks that are a few weeks old, especially if the birds are immunosuppressed with chicken infectious anaemia virus, infectious bursal disease and other non-infectious agents (Toro *et al*., 2001; Shivachandra *et al*., 2003). This study was conducted to know the prevalence of concurrent infections such as MDV, ALV, REV and CIAV in the fowl adenovirus FAdV positive samples.

***Corresponding author E-mail: chitradevi.dr@gmail.com**

MATERIALS AND METHODS

The samples were collected from fowl adenovirus suspected commercial broiler birds between the age groups of 2 to 6 weeks in Tamil Nadu from November 2016 to December 2017.

Liver tissues were collected from affected flock and stored at -80°C. DNA was extracted from liver tissues using DNeasy Tissue Kit (QIAGEN) as per the manufacturer's protocol. The concentration and purity of the extracted DNA was checked by Nanodrop. PCR was carried out directly from the DNA extracted from liver samples and for PCR the following primer sequences were used in this study (Table 1)

RESULTS AND DISCUSSION

The present study was carried out to find out the concurrent viral infection in commercial broiler flocks

Table 1: Sequences primers used in this study

S.No	Disease	Primer	Amplicon size	Reference
1.	ALV	ALV-FP-5'ACG GAT TTY TGC CTY TCT3' RP- 5' ATT GTG YCT RTC CGC TGT C 3'	466bp	Ottiger, 2010
2.	CAV	CAV-FP-5'CTAAGATCTGCAACTGCGGA 3' CAV-RP-5' CCTTGGAAGCGGATAGTCAT 3'	419bp	Ottiger, 2010
3.	MDV	MDV-F-5'GTCCCCCCTGATCTTTCTC3' MDV-R-5'CGTCTGCTTCCTCGTCTTC3'	184bp	Abdul-Careem *et al*. (2006)
4.	REV	REV-FP-5' CAT ACT GGA GCC AAT GGT T 3' REV-RP-5' AAT GTT GTA CCG AAG TAC T 3'	291bp	Ottiger, 2010

which were confirmed with fowl adenovirus infection. Affected flocks showed dullness, reduced feed intake, ruffled feather and mortality recorded in that affected flock was 6 to 20 per cent. On postmortem examination enlarged pale yellow and friable liver congested kidney and ascites were observed.

Out of 27 FAdV positive flocks, 16 flocks (59%) were found positive for CIAV in VP2 gene PCR (Fig. 1). Co infection of CIAV and FAdV in 20 day old chickens with intramuscularly showed 55% mtitis ortality and produced characteristic signs and lesions of inclusion body hepatitis/hydopericardium syndrome (IBH/HPS) whereas, birds singly infected with FAdV showed 10% mortality due to IBH/HPS (Toro *et al*., 2000) but in this study 6 to 20 per cent mortality was recorded. Immunosuppression before or concurrently with FAdV infection serves as an important factor for developing clinical presentations, such as IBH and HPS (Choi *et al*. 2012). Association of FAdV and CAV is necessary for the successful induction of the IBH/HP syndrome in chickens when transmitted vertically (Toro *et al.* 2001).

Out of 27 FAdV positive flocks, 21 flocks (77%) were found positive by PCR for 466 bp of ALV envelope gp85 (Fig. 2). Sathish *et al.* (2015) reported the presence of ALV subgroup E in commercial layer birds by PCR. Elamurugan *et al.* (2014) screened and confirmed the presence of ALV-A infection in laying hens and their embryos. Presence of endogenous ALV can have direct or indirect effects on immune responsiveness and performance (Crittenden, 1991; Gavora *et al*., 1991). Nine flocks (33%) were found positive for both CIAV and ALV. Aly *et al*. (2004) reported dual infection of ALV

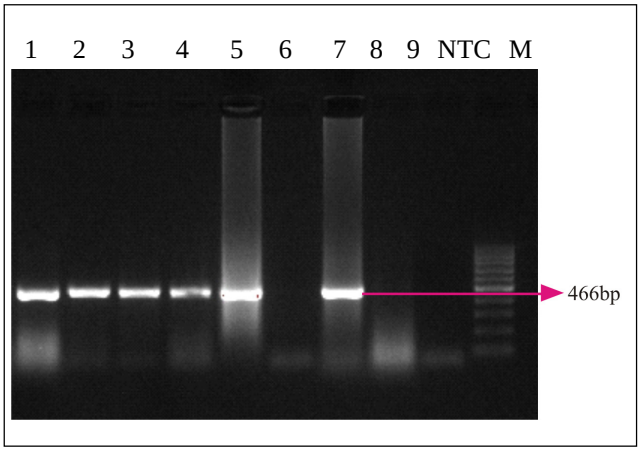

Fig. 1: Agarose gel electrophoresis of envelope gene (gp85) of ALV
(From lane 1 to 9 samples, NTC-No template control and M-100 bp DNA ladder)

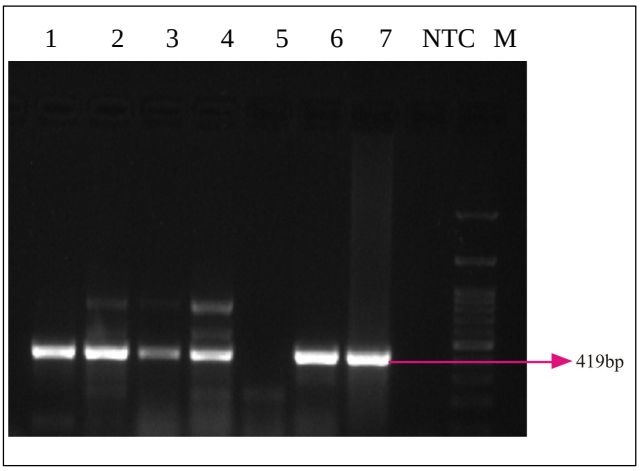

Fig. 2: Agarose gel electrophoresis of Vp2 gene of CIAV
(From lane 1 to 7 samples, NTC-No template control and M – 100 bp DNA ladder)

-J and CIAV in commercial broiler chicken affected with stunting syndrome. No MDV and REV positives were recorded in this study.

CONCLUSION

Most clinical cases of FAdV infection in the present study were co-infected by immunosuppressive viral agents such as CIAV and ALV. Control methods for immunosuppressive diseases mainly by minimizing stress, reducing exposure to infectious agents through proper biosecurity measures and increasing host resistance to infectious immunosuppressive diseases by vaccination should be implemented. Interaction and synergistic effects of viruses and viral genes also to be studied for further prevention of infection.

ACKNOWLEDGEMENTS

Authors thank the Dean, Veterinary College and Research Institute, Namakkal, Tamil Nadu Veterinary and Animal Sciences University for providing facilities to carry out the work.

REFERENCES

Abdul-Careem, M.F., Hunter, B.D., Nagy, E., Read, L.R., Sanei, B., Spencer J.L., Sharif, S. (2006). Development of a real-time PCR assay using SYBR Green chemistry for monitoring Marek's disease virus genome load in feather tips. *J. Virol. Methods*, 133: 34-40.

Aly, M.M., Hassan, M.K., Lueschow, D., Hafez, H.M. (2004). Stunting syndrome associated with Avian Leukosis Virus subgroup J and Chicken Infectious Anaemia in broiler flocks in Egypt. *Archiv fur Geflugelkunde*, 68: 57-61.

Choi. K.S., Kye, S.J.J., Kim, Y., Jeon, W.J., Lee, E.K., Park, K.Y., Sung, H.W. (2012). Epidemiological investigation of outbreaks of fowl adenovirus infection in commercial chickens in Korea. *Poult. Sci. J.*, 91: 2502–2506.

Crittenden, L.B. (1991). Retroviral elements in the genome of the chicken: Implications for poultry genetics and breeding. *Cri. Rev. Poult. Biol.*, 3: 73-109.

Dahiya, S., Srivastava, R.N., Hess, M., Gulati, B.R. (2002). Fowl adenovirus serotype 4 associated with outbreaks of infectious hydropericardium in Haryana. *Avian Dis*., 46: 230-233.

Elamurugan, A., Tomar, A., Saxena, V.K. (2014). Identification of True Exogenous Avian Leukosis Virus Transmitters by Serological and Molecular Assays. *J. Pure Appl. Microbiol.,* 8: 3299-3307.

Gaba, A., Parmar, H., Pal, J.K., Prajapati, K.S. (2010). Isolation, identification and molecular characterization of Inclusion body hepatitis virus. *Vet. World.,* 3: 415-417.

Gavora, J.S., Kuhnlein, U., Crittenden, L.B., Spencer, J.L. Sabour, M.P. (1991). Endogenous viral genes: Association with reduced egg production rate and egg size in White Leghorns. *Poultry Sci.,* 70: 618-623.

Kumar, R., Chandra, R., Shukla, S.K. (2003). Isolation of etiological agent of hydropericardium syndrome in chicken embryo liver cell culture and its serological characterization. *Indian. J. Biol.*, 41: 821-826.

McFerran, J.B. and Adair, B.M. (2003). Group 1 adenovirus infections. In: Saif, Y., Fadly, A., Glisson, J., Mcdougald, L., Nolan, L. and Swayne, D. (Eds). Diseases of Poultry. 12th Ed., Blackwell Publishing, Iowa State University Press, USA, Ames. pp 214–227.

McFerran, J.B., Smyth, J.A. (2000). Avian adenoviruses. *Rev. Sci. Tech.,* 19: 589– 601.

Ottiger, H.P. (2010). Development, standardization and assessment of PCR systems for purity testing of avian viral vaccines. *Biologicals*., 38: 381-388.

Sathish, G., Parthiban, M., Divya K.C., Kumanan, K., Vedamanickam, S. (2015). Detection of Avian Leukosis Viruses in commercial poultry flocks by Immunohistochemistry and PCR. *Int. J. Adv. Res. Biol. Sci*., 2: 215–224.

Shivachandra, S.B., Sah, R.L., Singh, S.D., Kataria, J.M., Manimaran, K. (2003). Immunosuppression in broiler chicks fed aflatoxin and inoculated with fowl adenovirus serotype 4 associted with hydropericardium syndrome, *Vet. Res. Comm.,* 27: 39-51.

Toro, H., Gonzalez, C., Cerda, L., Hess, M., Reyes, E., Geisse, C. (2000). Chicken anemia virus and fowl adenoviruses: association to induce the inclusion body hepatitis/hydropericardium syndrome. *Avian Dis.,* 44: 51–58.

Toro, H., González, O., Escobar, C., Cerda, L., Morales, M.A., Gonzalez, M.A. (2001). Vertical induction of the inclusion body hepatitis/hydropericardium syndrome with fowl adenovirus and chicken anemia virus. *Avian Dis.*, 45: 215-222.