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Fowl Adenovirus Induced Gizzard Erosion in Broiler Chickens in Southern India

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ABSTRACT

Background: In Southern India, feed passage syndrome was on the rise in the late half of 2019 affecting broiler chicken flocks. The feed passage syndrome is an indication of poor gastrointestinal health in chickens, in which poorly digested coarse feed particles are observed to be more in the feces. This study investigated the causative factors for the feed passage syndrome in broiler flocks.

Methods: Affected commercial broiler farms having incidences of feed passage syndrome, low body weight gain and poor feed conversion ratio, were visited in several regions of Karnataka and Andhra Pradesh. But there was no increase in mortality. Feed samples were collected from these regions for mycotoxin analysis. Intestinal health was examined in these flocks for lesions of coccidiosis and bacterial enteritis/dysbacteriosis. The common observation in all the birds examined was the presence of gizzard erosions. The gizzard in most of the birds was flaccid and moderate to severe erosions in the koilin layer of the gizzard were observed. In some birds, there were mild to moderate lesions caused by *Eimeria maxima* and *Eimeria tenella*. Tissue impressions on FTA cards were collected from gizzards having erosion on the koilin layer and submitted for identification of Fowl Adenovirus (FAdV) through the polymerase chain reaction (PCR) method.

Result: All the samples were found positive for FAdV. Four samples were further processed for sequencing of the Hexone gene to identify the serotype of the virus. FAdV serotype 11 was detected in three samples and serotype 8a was detected in one. Among major mycotoxins, low levels of Aflatoxin B1 were the predominant finding in feed samples. Thus, it can be concluded that fowl adenovirus-induced gizzard erosions in commercial broiler birds along with mild coccidiosis could be leading to feed passage syndrome.

Key words: Broiler chicken, Feed passage syndrome, Fowl adenovirus, Gizzard erosion, Phylogenetic analysis, Sequencing.

INTRODUCTION

With rising feed ingredient prices, feed conversion ratio (FCR) plays an important role in deciding the economics of commercial broiler farming. Efficient feed conversion ensures maximum utilization of feed for converting into body mass. The gastrointestinal tract (GIT) plays a vital role in the digestion and absorption of nutrients. Several factors related to feed quality and infections can affect GIT thereby hampering the digestion and absorption processes.

Digestive disorders in broiler chickens are commonly observed throughout India. The severity of digestive disorders increases during certain seasons, especially summer and monsoon. Poor body weight gain and flock uniformity are the common findings in such affected flocks. In most cases of GIT disorders, coccidiosis is found to be the major causative factor. Seven species of coccidia have been found to infect chicken. Among the seven coccidia species, *Eimeria acervulina*, *Eimeria maxima* and *Eimeria tenella* have significant economic importance (Thenmozhi *et al*., 2014). These three species of coccidia are most found to affect broilers. *Eimeria acervulina* and *Eimeria maxima* cause lesions in the small intestine, whereas *Eimeria tenella* causes pathologic changes in the ceca. Diagnosing coccidiosis is relatively easy by just gross examination of organs for lesions and detecting coccidia oocysts in birds' feces (Fatoba *et al*., 2018). But in other cases, apart from coccidiosis, it becomes unclear to diagnose just by gross postmortem examination. Due to the lack of sufficient diagnostic facilities, it becomes difficult to diagnose the exact factor causing digestive disorders. The condition of feces can give an idea of the status of birds' intestinal health.

The feed passage syndrome is one such condition affecting the gastrointestinal health of chickens, in which poorly digested coarse feed particles (especially maize in a corn-based diet) are observed to be more in the feces. Several factors related to feed quality as well as infectious causes can predispose broiler birds to feed passage syndrome. The presence of feed passage syndrome is an indication of poor intestinal health of the birds, leading to poor feed conversion and body weight gain (Butcher *et al*., 2002).

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The gizzard is an important part of avian GIT, which is a muscular organ located between the proventriculus and duodenum. The major function of the gizzard is to grind coarse feed particles ingested into finer ones to facilitate the digestion of ingesta (Svihus, 2014). This role of the gizzard becomes vital in birds due to the lack of the mastication mechanism. The mucosal layer of the gizzard is lined by a smooth and tough layer of a carbohydrate-protein complex called the koilin layer (Gjevre *et al*., 2013). The koilin layer helps to protect the delicate gizzard mucosa from abrasions during the grinding process (Svihus, 2014). Any abnormality or erosion on the koilin layer is commonly termed 'gizzard erosion'. Several factors are known to cause gizzard erosion (GE). Mycotoxins, biogenic amines, gizzerosine, copper sulfate, *Clostridium perfringens*, Fowl Adenovirus, etc. are the major ones causing GE in chicken (Gjevre *et al*., 2013). All these factors causing GE also can have deleterious effects on birds' intestinal integrity. Poor intestinal integrity can lead to poor feed conversion and a drop in flock performance.

High virulent serotypes of Fowl Adenovirus (FAdV) are commonly known to cause inclusion body hepatitis (IBH) in chickens, especially young broilers leading to an increase in mortality which may go up to 30% (Swayne *et al*., 2013). A less virulent form of FAdV may not cause IBH-like lesions or a drastic increase in mortality but can affect the bird's health and performance. Less virulent strains of FAdV are known to affect other organs like the lungs, heart, pancreas and gizzard, causing respiratory disease, hydropericardium syndrome, necrotizing pancreatitis and GE respectively (Balamurugan *et al*., 2004; Schade *et al*., 2013; Dhillon *et al*., 1982; Manarolla *et al*., 2009; Ono *et al*., 2001 and Tanimura *et al*., 1993). In one case involving spiking mortality syndrome in broiler chicks, FAdV has been detected in the pancreas, small intestine and liver, causing hypoglycemia in birds (Goodwin *et al*., 1993). In cases of GE caused by FAdV, macroscopic changes evident during gross examination are erosions on the koilin layer and inflammation or ulcers on the mucosal surface of the gizzard (Grafl *et al*., 2013). Microscopic examination of gizzard revealed degenerated glandular epithelial cells with intranuclear infiltrations, along with inflammatory cell infiltration of the lamina propria, submucosa and muscle layer of the gizzard (Grafl *et al*., 2013). This suggests that the virus causes damage to glandular epithelial cells leading to abnormalities in the koilin layer as well as other tissues of the gizzard as well. Studies have also been conducted to confirm the infectivity and involvement of FAdV in causing GE in chickens. In one such study, after isolating the FAdV strain from birds affected with GE, the inoculate with the isolated virus was administered to the healthy broilers. The inoculated birds were euthanized and examined through necropsy and histopathologic examinations. The inoculated birds upon necropsy, exhibited degenerative koilin layer. Microscopic examination of the gizzard revealed necrotic mucosa, intranuclear inclusion bodies in the glandular epithelial cells, inflammatory cell infiltrations in the lamina propria, submucosa and muscle of the gizzard. Intranuclear inclusion bodies of epithelial cells in the gizzard, ileum and cecal tonsil were further processed for immunohistochemical staining and FAdV antigen was identified in it (Ono *et al*., 2003). Cases of FAdV-associated GE have been observed in several countries (Schade *et al*., 2013; Bulbule *et al*., 2016; Manarolla *et al*., 2009 and Ono *et al*., 2001). In India, most of the work for the identification and genotyping of FAdV in cases involving GE in chicken has been done only in layers (Bulbule *et al*., 2016 and Chitradevi *et al*., 2020). In the case of GE, the affected birds will not show any specific clinical signs and may go unnoticed by untrained personnel. GE-affected birds were found to be associated with growth retardation (Mirzazadeh *et al*., 2021). Since several factors can have deleterious effects on the GIT of the birds, it becomes imperative to identify the exact causative factor to take necessary preventive measures.

During the latter half of 2019, incidences of feed passage syndrome were observed to be on the rise in Karnataka and Andhra Pradesh. Affected commercial broiler farms having incidences of feed passage syndrome were visited in several regions of Karnataka *viz.* Bengaluru, Mangalore and Chikmagaluru and Chittoor in Andhra Pradesh. Intestinal health analysis was done in affected flocks to check for coccidiosis, bacterial enteritis and overall health of the gastrointestinal tract. The study reported herein was conducted to investigate the causative factors responsible for feed passage syndrome in commercial broiler chicken flocks in these regions.

CASE REPORT

History of the flocks

A drop in broiler performance and an increase in feed passage syndrome were reported in several regions in Southern India. The broiler flocks were affected from the third week onwards. Twenty-eight farms of various age groups having incidences of feed passage syndrome were visited in Bengaluru, Mangalore, Chikmagaluru and Chittoor regions in southern India from June to October 2019. All these broiler flocks were from different breeder flocks and none of them were vaccinated against FAdV. The vaccination status of breeder flocks against FAdV from which these broiler flocks were is unknown. Details of raw materials used for feed manufacturing were collected. In each flock, farm conditions, bird behavior and health, performance data and farm management practices were examined.

Intestinal health examination

For investigating causative factors leading to GIT disturbances, in each farm, three live birds were randomly sampled and euthanized humanely and the gastrointestinal tract was examined. GIT was thoroughly examined for lesions of coccidiosis (*Eimeria acervulina*, *Eimeria maxima* and *Eimeria tenella)* and bacterial enteritis/dysbacteriosis.

Coccidiosis lesions were scored and recorded based on severity according to the method of Johnson and Reid (Johnson *et al*., 1970), whereas dysbacteriosis lesions were scored based on the method by Teirlynck *et al.* (Teirlynck *et al*., 2011).

Samples collected

To rule out feed-related factors, in this case, mycotoxins, twenty-five feed samples were collected from the affected farms and sent to the laboratory for the following mycotoxin analysis: Aflatoxin B1, Ochratoxin A, Citrinin, Trichothecene (T2) and Zearalenone. For identification of FAdV in affected flocks, during intestinal health examination, tissue impression on FTA card was collected from gizzards having erosions on the koilin layer. Six such gizzard impression samples were collected from these regions and submitted to the Department of Veterinary Microbiology, Veterinary College and Research Institute, Namakkal, for identification of FAdV through the polymerase chain reaction (PCR) method. Four positive samples were further processed for sequencing of the Hexon gene of FAdV to identify the serotype of the virus.

OBSERVATIONS

In all the farms, feed passage (undigested maize particles in birds' feces) was observed throughout the house (Fig 1). Along with feed passage, orange-colored mucus in the feces was also observed in several farms. There was no significant increase in mortality. In all the flocks, after the feed passage started, a decline in weekly body weight gain was observed and was lower than the breed standards. During the examination of GIT, the presence of gizzard erosion was the most common observation in all the birds. Gizzards from most of the birds appeared flaccid with reduced musculature and moderate to severe erosions in the koilin layer of the gizzard were observed (Fig 2). Proventriculus was found to be normal. In a few birds, mild to moderate *Eimeria maxima* (Fig 3) and *Eimeria tenella* lesions were observed. During intestinal health examination, poorly digested maize particles were observed in the content of the distal ileum.

Low body weight gain and poor feed conversion were common observations in all the flocks examined. But there was no increase in mortality. During intestinal health examination, gizzard erosion was the most common observation. GE was observed in 100% of the birds examined for GIT assessment. Affected gizzards had reduced musculature and appeared flaccid. Coccidiosis was the second most observed condition affecting GIT and was observed in 57% of the birds examined for GIT assessment. Among coccidiosis-positive birds, 60% of the birds had scored 1 (mild) lesion whereas the rest 40% had scored 2 (moderate) lesions. Among coccidiosis lesions, *E. maxima lesions* were predominant. However, the overall mean lesion score was found to be on the lower side (Fig 4). The dysbacteriosis lesion scores were found to be on the higher side in most of the regions, except in Chittoor (Fig 5).

The feed provided to all these flocks was formulated with plant-origin ingredients. Raw materials from animal protein sources were not used in the feed. Out of twenty-five feed samples collected, Table 1 shows the number of samples collected from each region. In the feed samples submitted for mycotoxin analysis, the limit of quantification was 8ppb for all the mycotoxins tested. Aflatoxin B1 was detected as the most prevalent mycotoxin and was found in all the feed samples. The level of aflatoxin B1 detected ranged from 8 to 54 ppb (Table 2). In all the feed samples, ochratoxin was either not present or was below the limit of



Fig 1: Feed passage (Undigested feed particles in feces). IMG-KAI-00165/00166.



Fig 2: Erosions on koilin layer of gizzard. IMG-KAI-00167/00168/00169/00170.

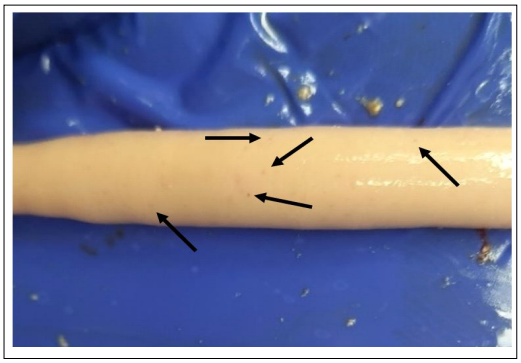


Fig 3: *Eimeria maxima* lesions (petechial hemorrhages on the serosal surface of the small intestine) - IMG-KAI-00171.

quantification (Table 3). Citrinin was detected in only two out of twenty-five feed samples. In the rest of the feed samples, citrinin was either not present or was below the limit of quantification (Table 4). Zearalenone was detected in two feed samples. In the rest of the feed samples, zearalenone was either not present or was below the limit of quantification (Table 5). Trichothecene was also either not present or was below the limit of quantification (Table 6) in all the feed samples submitted to the laboratory for mycotoxin analysis.

All six gizzard impression smear samples submitted for the detection of FAdV by PCR were found positive for FAdV (Table 7). Four of these positive samples were further processed for sequencing of the Hexone gene of FAdV. Phylogenetic analysis of the gene fragment indicated a resemblance to fowl adenovirus serotype 11 in three samples and serotype 8a in one sample (Table 7 and Fig 6, 7, 8 and 9).

Table 1: Different numbers of feed samples were collected (region-wise).

Region	Number of feed samples collected
Bangalore	12
Mangalore	1
Chikmagaluru	4
Chittoor	8

DISCUSSION

A Gizzard erosion is a syndrome caused by multiple etiological factors. Broadly these factors can be grouped into two; feed related and infectious (Gjevre *et al*., 2013).

Table 2: Aflatoxin B1 levels detected in feed samples (Unit: ppb).

Region	Sample number											
	1	2	3	4	5	6	7	8	9	10	11	12
Bangalore	8	8	8	8	8	8	8	12	12	17	20	24
Mangalore	40	-	-	-	-	-	-	-	-	-	-	-
Chikmagaluru	40	48	54	54	-	-	-	-	-	-	-	-
Chittoor	8	16	20	24	24	24	32	32	-	-	-	-

'- ': No sample.

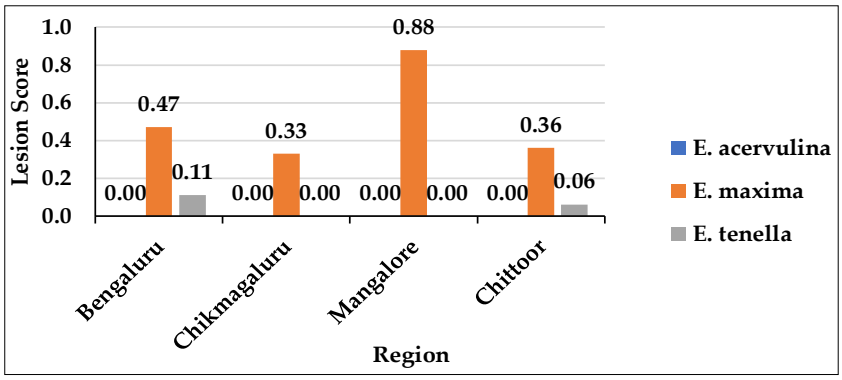


Fig 4: Coccidiosis lesion scores.

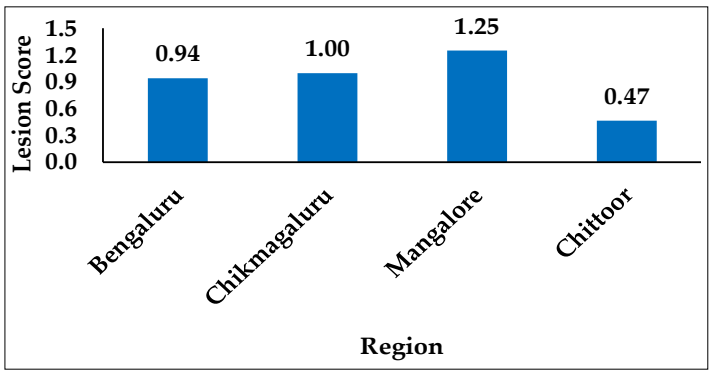


Fig 5: Dysbacteriosis lesion scores.

Among feed-related factors, biogenic amines, gizzerosine and mycotoxins are the major ones (Gjevre *et al*., 2013). Biogenic amines present in poorly processed animal protein sources have been shown to cause GE in broilers (Barnes *et al*., 2001). Gizzerosine present in fish meal has also been found to cause GE in chicken (Masumura *et al*., 1985). In the present study, feed provided to all the examined flocks did not have any animal protein sources. So, chances of biogenic amines and gizzerosine as a causative factor for GE were ruled out. Among mycotoxins, those belonging to the trichothecenes group, especially T2, deoxynivalenol (DON), fumonisins and cyclopiazonic acid (CPA) are potent

Table 3: Ochratoxin A levels detected in feed samples (Unit: ppb).

Region	Sample number											
	1	2	3	4	5	6	7	8	9	10	11	12
Bangalore	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8
Mangalore	<8	-	-	-	-	-	-	-	-	-	-	-
Chikmagaluru	<8	<8	<8	<8	-	-	-	-	-	-	-	-
Chittoor	<8	<8	<8	<8	<8	<8	<8	<8	-	-	-	-

'- ': No sample.

Table 4: Citrinin levels detected in feed samples (Unit: ppb).

Region	Sample number											
	1	2	3	4	5	6	7	8	9	10	11	12
Bangalore	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8
Mangalore	40	-	-	-	-	-	-	-	-	-	-	-
Chikmagaluru	20	<8	<8	<8	-	-	-	-	-	-	-	-
Chittoor	<8	<8	<8	<8	<8	<8	<8	<8	-	-	-	-

'- ': No sample.

Table 5: Zearalenone levels detected in feed samples (Unit: ppb).

Region	Sample number											
	1	2	3	4	5	6	7	8	9	10	11	12
Bangalore	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8
Mangalore	<8	-	-	-	-	-	-	-	-	-	-	-
Chikmagaluru	102	210	<8	<8	-	-	-	-	-	-	-	-
Chittoor	<8	<8	<8	<8	<8	<8	<8	<8	-	-	-	-

'- ': No sample.

Table 6: Trichothecene (T2) levels detected in feed samples (Unit: ppb).

Region	Sample number											
	1	2	3	4	5	6	7	8	9	10	11	12
Bangalore	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8	<8
Mangalore	<8	-	-	-	-	-	-	-	-	-	-	-
Chikmagaluru	<8	<8	<8	<8	-	-	-	-	-	-	-	-
Chittoor	<8	<8	<8	<8	<8	<8	<8	<8	-	-	-	-

'-' : No sample.

Table 7: FAdV PCR and gene sequencing result.

Region	No. of samples submitted for PCR	No. of samples positive for FAdV	No. of samples submitted for gene sequencing	FAdV serotype detected
Bengaluru	3	3	2	11
Mangalore	1	1	Nil	Nil
Chikmagaluru	1	1	1	11
Chittoor	1	1	1	8a

in causing GE (Gjevre *et al*., 2013). In the feed samples collected from the study regions, Trichothecene was not detected in any of the feed samples. Other mycotoxins known to cause GE (DON, CPA and fumonisins) were not assessed in these feed samples.

Among infectious factors, the major ones are *Clostridium perfringens* and FAdV (Gjevre *et al*., 2013). In most incidences of GE, FAdV has been detected in affected birds (Schade *et al*., 2013; Bulbule *et al*., 2016; Manarolla *et al*., 2009 and Ono *et al*., 2001). While adenoviruses have been detected even in healthy chickens, certain pathogenic strains of the FAdV significantly affect birds' health and performance (Yates *et al*., 1976). The status of the immune system of the birds also plays a key role in deciding the severity of FAdV infection. Immunocompromised birds are found to have a severe manifestation of FAdV infection (Fadly *et al*., 1976). Less pathogenic strains of FAdV may not cause a significant increase in mortality but will affect flock performance. Gizzard erosion is such a kind of FAdV manifestation where clinical symptoms may not be apparent and mortality may not increase significantly, but body weight gain and feed conversion are affected (Mirzazadeh *et al*., 2021). FAdV serotype 1 has been identified in most of the cases involving GE globally (Mirzazadeh *et al*., 2019; Schade *et al*., 2013; Domanska-Blicharz *et al*., 2011; Manarolla *et al*., 2009; Ono *et al*., 2001; Ono *et al*., 2003). FAdV serotypes 8 and 11 were isolated from birds having GE in Japan and Iran (Mirzazadeh *et al*., 2019; Okuda *et al*., 2004). In India, FAdV serotypes 2, 3, 4, 8 and 11 were identified from cases involving GE in commercial layer chicken (Bulbule *et al*., 2016; Chitradevi *et al*., 2020). All the tissue impression samples collected for this study were found to be positive for FAdV. Thus, correlating with the findings of other workers, indicating the involvement of FAdV in causing GE in broiler chicken. In the present study, FAdV serotypes 8 and 11 were detected in the tissue impression samples contrary to the finding of FAdV serotype 1 in most of the studies. In cases of FAdV-induced GE, it has been demonstrated that the disease has an economic impact on the affected flock in terms of impaired body weight gain and an increase in mortality (Mirzazadeh *et al*., 2021). In the present study, though there was no significant increase in mortality, all the affected flocks had lower body weight gain than the breed standards.

Like GE, feed passage syndrome (FPS) is also caused by multiple etiological factors and FAdV is one of them (Butcher *et al*., 2002). Any condition or factors affecting GIT will impair the digestion process leading to the excretion of poorly digested feed particles in the feces. FAdV has been identified from GIT of broilers having feed passage problems (Apple *et al*., 1991). A similar observation was found in the present study as well. Feed passage was observed in all the farms visited with varying severity. Coccidiosis and dysbacteriosis lesions were also observed in most of the birds examined for intestinal health assessment. So, it could be interpreted that along with FAdV, coccidiosis and

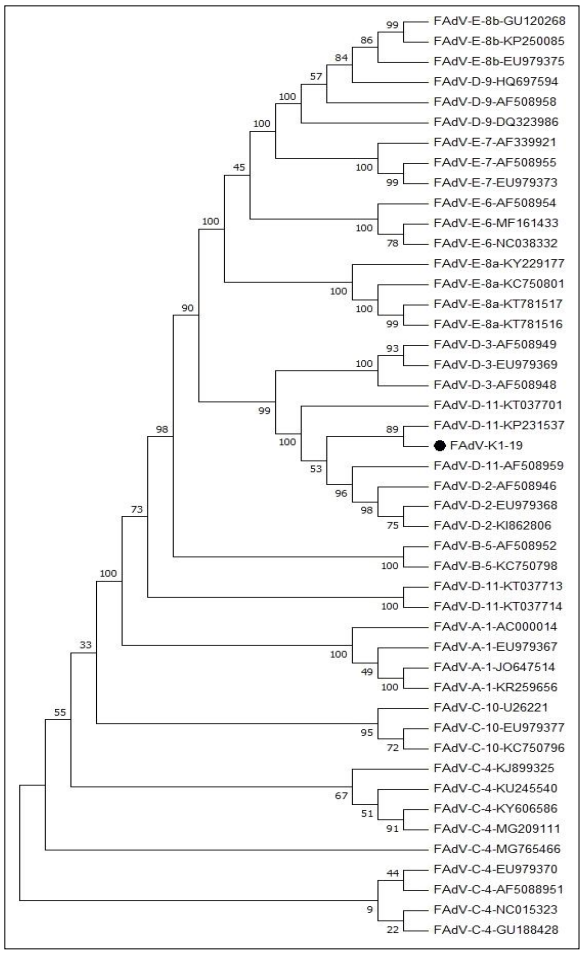


Fig 6: Phylogenetic analysis of sequenced Hexone gene of FAdV positive sample K1-19. The sample was similar to FAdV serotype 11.

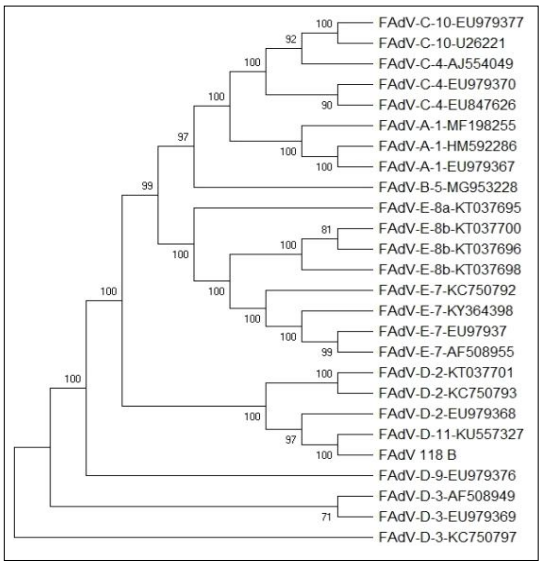


Fig 7: Phylogenetic analysis of sequenced Hexone gene of FAdV positive sample 118B. The sample was similar to FAdV serotype 11.

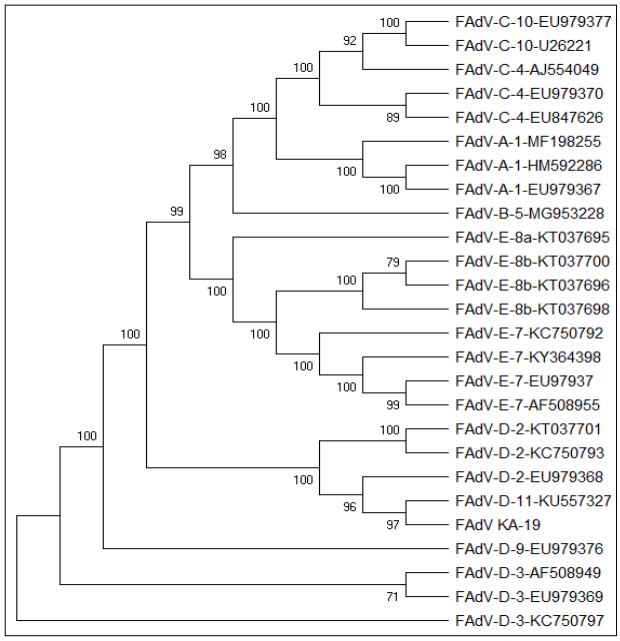


Fig 8: Phylogenetic analysis of sequenced Hexone gene of FAdV positive sample KA-19. The sample was similar to FAdV serotype 11.

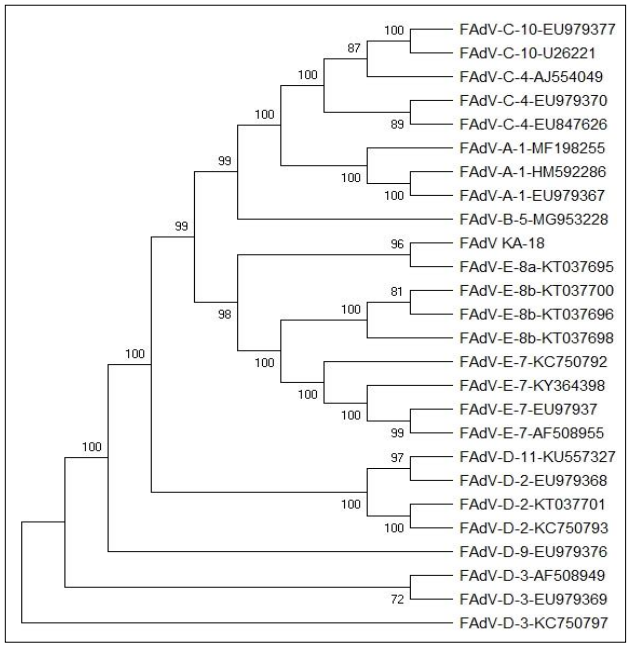


Fig 9: Phylogenetic analysis of sequenced Hexone gene of FAdV positive sample KA18. The sample was similar to FAdV serotype 8a.

dysbacteriosis can also be the contributing factors for the feed passage syndrome in affected flocks. The abuse caused by these factors to the intestinal integrity could be the reason for the poor digestion process leading to feed passage syndrome in commercial broiler flocks and poor performance parameters.

CONCLUSION

Feed passage syndrome is a complex phenomenon and can involve several etiological factors. Identifying the predisposing factors is vital for effectively addressing the feed passage syndrome. In the present study, gizzard erosions were the most predominant findings in all the flocks examined. FAdV serotypes 8 and 11 were found to be the causative factor for GE in the broiler flocks. Though T2 mycotoxin was not found in any of the feed samples, the probability of other mycotoxins causing GE (DON, fumonisins and CPA) cannot be ruled out as they were not tested. Feed passage syndrome in this case appears to be due to the combined effect of FAdV, coccidiosis and dysbacteriosis. Among these factors, pointing out the most impactful one in causing feed passage is difficult, as a histopathological examination was not conducted. A holistic approach is needed to prevent and control the feed passage syndrome in commercial broiler birds. In this case, vaccination against fowl adenovirus along with good anticoccidial and intestinal health management programs in the feed can help in reducing feed passage syndrome, leading to improved performance parameters in commercial broiler flocks.

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Conflict of interest

The authors have no conflict of interest.

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