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Short Communication

Technical report on laboratory outbreak investigation of sudden death syndrome in broiler chicken in Kathmandu valley Nepal 2009

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The incidence of death of broiler birds above 40 days suddenly increased in the month of July to October 2009 in Kathmandu valley. Birds that were presented for post-mortem examination at the Central Veterinary Laboratory Tripureswor Kathmandu were usually found dead on their backs with wings out-stretched. Gross abnormalities recorded on post mortem examinations were: muscle oedema, pulmonary, renal and liver congestion, dark black to pale yellow streaked liver bile filled gall bladder and congestive splenomegaly, blood clot in atrium haemorrhage in duodenal muscle, whitish yellow pasty fluid in proventriculus gland, greenish coloration marked intact feed particles in gizzard and excessive mucous filled swollen intestine. Incidence rate was recorded between 1.5 to 2.5% of the flock. The mean mortality rate due to sudden death syndrome was 1.3 to 9.6%. *Penicillium* spp. *Aspergillus* spp. with CFU ranging from 56 × 10^4 to 62 × 10^5, to uncountable mold count ' *Escherichia coli, Streptococcus* spp. and *Staphylococcus* spp. were the usual organisms isolated from culture samples of liver, lung, spleen and proventriculus. The condition seems to be related to mycotoxicosis. Reduction of mortality was achieved by feed restriction, with 8 to 10% reduction in nutrient density. Supplementation of glucose containing electrolyte, liquid toxin binder, immuno-modulator, acidifier and antibiotic therapy.

Key word: Sudden death syndrome, broiler birds, Kathmandu valley, *Penicillium*, CFU, ranging from 56 × 10^4 to 62 × 10^5 g, uncountable mold count.

INTRODUCTION

Sudden death syndrome (SDS) has been recognized for over 30 years, and is also referred to as acute death syndrome or "flip-overs". It is most common in males when their growth rate is maximized. Mortality may start as early as 3 to 4 days, but most often peaks at around 3 to 4 weeks of age, with affected birds being found dead on their back. Mortality may be found at 1.5 to 2.0% in mixed-sex flocks and as high as 4% in male flocks only (George, 2007). Sudden death syndrome has developed into a major problem to the broiler industry in many parts of the world. Broilers of all ages are affected starting as early as 2 days of age and continuing through to market age. Peak mortality usually occurs between 3 and 4 weeks of age (Gardiner et al., 1988). The syndrome has been reported to cause between 1.31 and 2.46% mortality with males more commonly affected than females (Riddell and Orr, 1980). However, Proudfoot et al. (1982) reported 0.90 to 3.61% mortality due to SDS in broilers. From 0.71 to 4.07% as reported by Riddel and Springer (1985) whereas, Ononiwu et al. (1979) reported 1.0% mortality due to SDS in broilers. Brigden and Riddell (1975) reported that 70 to 80% of male mortality and 20 to 25% of female broilers chickens mortality could be attributed to acute death syndrome or "Flip-Over Disease". Thus, SDS is a leading contributor to mortality in broiler chicken production. Although, the precipitating event is yet to be ascertained," Cardiovascular failure" appears to be the immediate cause of death (Siddiqui et al., 2009).

OUTBREAK OF SUDDEN DEATH SYNDROME IN KATHMANDU VALLEY

From the first week of July to October 2009, there was a sudden increase in mortality of broilers above 6 weeks of

Table 1. Epidemiology of affected flock with sudden death syndrome in July to October, 2009.

Observation/duration	No.of farmers/flock	Population at risk	Morbidity (%)	Mortality (%)	No. of samples examined
July	63	16620	4250 (25.57)	369(2.22)	63
August	51	15450	1235 (7.99)	232 (1.50)	51
September	32	10260	848 (8.26)	157 (1.53)	32
October	30	15700	2380 (15.16)	149 (0.94)	30
Total	176	58030	8713 (15.01)	907 (1.56)	176

Table 2. Results of microbiological examination.

No. of samples	Bacterial isolated	Fungi isolated	Positive no.	Negative no.
176	*Escherichia coli* *Streptococcus* *Staphylococcus*		35	141
176		*Aspergillus* *Penicillium*	145	31

Table 3. Rapid test for AI, ND, IBD.

Variable	No. of samples	Positive	Negative
AI	20	0	20
ND	20	o	20
IBD	20	o	20

age (Table 1) in Kathmandu valley. There were no premonitory signs. Just before death, birds appear normal and it is common to observe the birds if they eat, drink or walk normally. Then birds use to exhibit clinical signs such as extending their neck, squawk and start wing beating as well as, leg extension before falling on their back.

POSTMORTEM FINDING OF SDS BIRDS

Gross abnormalities recorded on post mortem examinations were muscle oedema, pulmonary, renal and liver congestion, dark black to pale yellow streaked liver bile filled gall bladder and congestive splenomegaly, blood clot in atrium haemorrhage in duodenal muscle, whitish yellow pasty fluid in proventriculus gland, greenish coloration marked intact feed particles in gizzard and swollen intestine with excessive mucous filled. All these post mortem observations conform to the descriptions of the syndrome made by Ononiwu et al. (1979).

Laboratory finding of Mycobiota and Microbiota of postmortem tissue samples

A total 176 tissue samples of lung, liver, spleen, proventriculus and gizzard, were collected during postmortem examination and were subjected for both bacterial and mycological culture. Results of microbiological examination are given in Tables 2 and 3.

Treatment and preventive measure given to the rest of birds in flock

All birds remaining in flocks were subjected to restricted feed up to 8 to 10% these percentages differ from the ones in the abstract, and fed twice daily. Supplementation with glucose containing electrolyte, liquid toxin binders, immunomodulator, and simple broad-spectrum antibiotics and acidifiers were provided in water. Vitamin B complex supplementation was totally withdrawn. All birds remaining in all affected farms responded well to the management and there was a marked improvement in the overall condition of the flock.

RESULTS AND DISCUSSION

Sudden death syndrome (SDS) is an acute heart failure disease that affects mainly fast growing male chickens that seem to be in good condition. Although, a common feature in fast growing birds is that the pathogenesis remains unclear (Ononiwu et al., 1979). Cardiac arrhythmias are involved in the pathogenesis of SDS with ventricular arrhythmias (VA) being the most common observation representing premature ventricular contractions and fibrillation (Olkowski and Classen, 1997; 1998). It has been reported that broilers fed with high vitamin D3 diet above the recommended levels in an attempt to prevent commonly occurring leg problems

were 2.5 fold more likely to succumb to acute heart failure and die of SDS (Nain et al., 2007). SDS was also experimentally induced by feeding diets containing the mycotoxin moniliformin that resulted into cardiac injury with subsequent alterations in cardiac electrical conductance (Reams et al., 1997) suggesting the possible role of chronic mycotoxicosis to the causation of SDS. Other implicated causes of SDS include continuous artificial lighting (Ononiwu et al., 1979b), deviations in dietary calcium and phosphorus (Scheideler et al., 1995), feeding crumble-pellet diets (Proudfoot et al., 1982), dietary fat content (Rotter et al., 1985) and feeding frequency (Bowes et al., 1988). The latter recommendation of restricted feeding supports well the previous observation that abdominal fat deposition increases the risk of SDS such that restriction on calorie: protein ratio decreases the incidence of SDS (Mollison et al., 1984).

CONCLUSION

The present investigation indicates that broilers in good body weight condition when not harvested timely and remaining in poultry shades for prolonged periods suffer stressful events and even die sudden. Also, it is possible that increased humidity and hot seasons favors the growth of mold and fungus in stored feeds increasing the risk of birds to mycotoxicosis. Detail Histopathological examination of affected organ need to be carried out for further verification of the involvement of mycotoxin for this syndrome.

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