

A Preliminary Clinical Laboratory Investigation of Endemic Spiking Mortality Syndrome of Broiler Chickens in Nepal

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Abstract

During the period of March-April-May 2008 first time a sudden and unexpected jump in mortality in Broiler of 8 to 16 days of age was reported from broiler farms from Chitwan and Kathmandu valley in Nepal. Affected birds become recumbent, depressed and often go into a star-gazing spasm. Those signs included, huddling of the birds, trembling, blindness, loud chirping, litter eating, ataxia, comatose, birds dead with breast down and feet and legs straight out behind birds. Death within two to six hours after the onset of the symptoms Postmortem Lesions found with this syndrome include hemorrhages in the liver with necrosis of liver cells, regressed thymus, regression of the bursa of Fabricius, dehydration with the accumulation of kidney urates, fluid in the crop, fluid in the lower gut and watery contents of the ceca. Yellow elastic shanks swollen joints. Molted appearance of brain. . The mortality lasted for three to five days, after which, the mortality patterns return to a relatively normal level. When treated with liquid toxin binders like toxol, toxolivum, livertonix like hepatocare, naturaliv, immunomodulators like immunocare, promin, pentasol and antibiotics there was check in mortality but the body weight recovery was not satisfactory only half as in comparison of in other illness. During this period laboratory culture of total 298 tissue specimen from dead bird was conducted which revealed growth of fungus spp like *Aspergillus* and *Penicillium* in 182 specimen while mixed *E. coli* and *Staphylococcus* were recovered in 68 specimen 24 specimen revealed the growth of *Salmonella* spp of bacteria and 24 samples were turn out to be negative while the attempt to isolate the Avian encephalomyelitis virus as it might be the cause suspected also turnout negative. On the basis of all laboratory findings and response to the treatment attempted finding of this preliminary investigation work is suggestive that the above syndrome indicates that mycosis emerging as one of new challenge for the early age broiler health management need to be thoroughly further investigated.

Keywords: Spiking Mortality Syndrome, Early Broiler chicken, Fungus, *Aspergillus*, *Penicillium*, Liquid Toxin binder, avian encephalomyelitis.

Objective of Study

1. To ascertain the cause of disease.
2. To Identify the causative agent of Disease.
3. To recommend the suitable treatment and prevention Methodes.

Materials and Methods

1. Epidemiological Study of outbreak.
2. Laboratory investigation of Postmortem samples.(Bacterial Culture, virus isolation, Fungal Culture).
3. Monitoring of treatment.

Observation of flock

Flock size	Age/days	Morbidity	Mortality
100-500			
(30)	7-14	35-100	20-30
500-1000			
(50)	8-21	100-300	40-100
1000-1500			
(130)	7-21	250-350	70-100
1500-2000			
(74)	6-18	350-450	230-350

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Laboratory investigation of Postmortem samples (Bacterial Culture, virus isolation, Fungal Culture)

Species	Total	positive	negative	Isolate fungus
Avian	298	182	24	<i>Penicillium</i> (110) <i>Aspergillus</i> (72) <i>Salmonella</i> (24) Mixed, <i>E.coli</i> & <i>Staphylococcus</i> (68)

Lesions observed during postmortem:

Haemorrhages in the liver with necrosis of liver cells, regressed thymus, regression of the bursa of Fabricius, dehydration with the accumulation of kidney urates, fluid in the crop, fluid in the lower gut and watery contents of the ceca. Yellow elastic shanks swollen joints. Molted appearance of brain.

Treatment advised: Affeted flocks were advised to treat with the broadspectrum antibiotics liquid toxinbinders like toxolivum, toxol along with vitamin B complex, livertonic, immunomodulator and electrolytes containing glucose for 7 day.

Results and Discussion

During the period of March April May 2008 first time a sudden and unexpected jump in mortality in Broiler of 8 to 16 days of age was reported from broiler farmer from Chitwan and Kathmandu valley in Nepal. The mortality lasts for three to five days, after which, the mortality patterns return to a relatively normal level. Affected birds become recumbent, depressed and often go into a star-gazing spasm. Those signs included, huddling of the birds, trembling, blindness, loud chirping, litter eating, ataxia, comatose, birds dead with breast down and feet and legs straight out behind birds. Death within two to six hours after the onset of the symptoms Postmortem Lesions found with this syndrome include hemorrhages in the liver with necrosis of liver cells, regressed thymus, regression of the bursa of Fabricius, dehydration with the accumulation of kidney urates, fluid in the crop, fluid in the lower gut and watery contents of the ceca. Yellow elastic shanks swollen joints. Molted appearance of brain. When treated with liquid toxin binders like toxol, toxolivum, livertonic like hepatocare, naturaliv, immunomodulators like immunocare, promin, pentasol like drugs there was check in mortality but the body weight recovery was not satisfactory only half as in comparison of in other illness. Laboratory culture of total 298 tissue specimen from dead bird revealed growth of fungus spp like *Aspergillus* and *Penicillium* in 182 specimen while mixed *E.coli* and *Staphylococcus* were recovered in 68 specimen 24 specimen revealed the growth of *Salmonella* spp of bacteria and 24 samples were turn out to be negative while the

attempt to isolate the Avian encephalo-myelitis virus as it might be the cause suspected turnout negative.

These Clinical Laboratory observation of the sudden illness and mortality pattern and postmortem findings and laboratory analysis of tissue, and treatment response support the findings of earlier workers. From approximately 1988 to 1990 the syndrome reached critical epidemic proportions particularly in the Delmarva area of the U.S. Isolated cases were also reported at that time in Georgia, Alabama and Arkansas. Approximately around 1992, During the first experiences with the disease a number of causative agents were implicated, yet the symptoms remained relatively the same. the incidence of spiking mortality in chickens took a severe jump in Georgia and to a lesser extent Alabama. Sporadic cases were also still being reported in the Delmarva/North Carolina area. Several possible causes were implicated, but nothing conclusive was identified. Since that time Dr. James Davis of the Georgia Poultry Laboratory, in conjunction with several researchers across the U.S., conducted exhaustive and extensive research and uncovered an emerging virus with other interesting revelations. Yet, the syndrome still occurs and no absolute eradication of the problem is in sight. The general symptom of spiking mortality in broilers is a sudden and unexpected jump in mortality from 8 to 16 days of age. The mortality lasts for three to five days, after which, the mortality patterns return to a relatively normal level. Affected birds become recumbent, depressed and often go into a star-gazing spasm. Death often occurs within two to six hours after the onset of the symptoms. Characteristically, birds that exhibit clinical symptoms but survive the acute phase, will continue to be unthrifty and stunted for the rest of the grow-out. The surviving birds may weigh as little as one half of the weight of unaffected flock-mates by the end of the grow-out cycle. The 1990 Delmarva Task Force on SMSC further defined the lesions and symptoms into Type A and Type B. Type A was defined as any house of birds experiencing a daily mortality of >5 birds per 1000 between 8 to 16 days of age. The symptoms must occur for duration of not more than three days, with birds exhibiting all or a portion of the physical signs and lesions characteristic of the syndrome. Those signs included, huddling of the birds, trembling, blindness, loud chirping, litter eating, ataxia, comatose, birds dead with breast down and feet and legs straight out behind birds. Males are predominantly affected and survivors exhibit great variation in sizes. Lesions described with this syndrome include hemorrhages in the liver with necrosis of liver cells, regressed thymus, regression of the bursa of Fabricius, dehydration with the accumulation of kidney urates,

fluid in the crop, fluid in the lower gut and watery contents of the ceca. Type B spiking was defined as any house of birds exhibiting a significant mortality during the ages of 8 to 16 days of age. The mortality may extend longer than three days but the rate is less than five deaths per 1000 per day. Also the Type B affected birds did not exhibit the symptoms and lesions of the Type A affected birds. Regardless of the "Type" of spiking mortality, one of the hallmark lesions is hypoglycemia or low blood sugar. Also most cases have been refractive to supplemental medication or feed changes. A number of causative agents have been implicated in this devastating syndrome. Mycotoxins or a microtoxin were first implicated. This was based on the evidence of liver lesions and trace-back examinations of affected flocks. Through trace-back investigations, several investigators found that affected farms received feed containing feed ingredients from specific geographic locations in the U.S. Further research at the University of Georgia indicated that the syndrome was indeed feed related. When the feed from affected birds was given to normal birds, the normal birds started exhibiting signs of SMSC. Management factors were suspected during the early outbreaks of SMSC by many live production managers. Several ideas were put forth such as cold half house brooding, starvation of the birds and starting chicks on old feed. Substantiation of these mismanagement situations are difficult to address. However, it is interesting that as some attention has been given to improving management practices, the incidence of SMSC has declined. Viruses were suspected during the initial outbreaks of SMSC. The isolation of an adenovirus from flocks experiencing the syndrome. An arenavirus has been consistently isolated, in extensive studies by Dr. James Davis, from the feces of chicks exhibiting SMSC. Dr. Davis' work has shown that higher numbers of arenavirus particles are found in the feces of broilers, broiler breeders and commercial layers experiencing enteritis. However, the feces of chickens experiencing spiking mortality, have particularly high numbers of virus particles. Since then, Dr. Davis and others have conducted a number of different experiments and have reproduced the syndrome with the arenavirus. However, it appears that it may take more than just a viral infection to incite the syndrome. Dr. Davis' research indicates that shortly after infection by the virus, a short period of starvation for the chicken is enough to elicit the syndrome. Arenaviruses are known to infect mice, other rodents, birds and men. The virus infects areas of the brain that regulate hormone levels in the body, particularly growth hormone. Mice infected with the arenavirus have been found deficient in growth hormone which results in

hypoglycemia and growth depression (Dr. Davis). As there have been a number of different causative agents suggested, there have been a number of suggested treatments and preventative measures. The only successful treatment of SMSC in the face of an outbreak has been the therapeutic use of quinolone antibiotic in the water. Other remedies tried have included, changing feed, vitamin supplementation in the water, and adding sugar to the drinking water. No consistent success have been achieved with these treatments. A number of management techniques have been employed to reduce the chance of SMSC. Some common-sense measures such as adequate warming of the post-brood chambers, rodent control and darkling beetle control appear to have offered some help. One technique that has offered the best help in reducing the incidence of the syndrome is a lighting program, which restricts the amount of light the broilers are exposed to. The theory of the lighting program is to increase the amount of darkness the broiler is exposed to as the bird matures. The effect of this is to increase the level of melatonin (a natural hormone released from areas of the brain). Higher levels of melatonin may help to elevate the level of growth hormone and reduce the opportunity for hypoglycemia. Thus far, it appears that the lighting program has been successful both experimentally and in practical usage. However, the lighting program has not proven to be a cure-all. Sporadic outbreaks of SMSC still occur regardless of what management practices have been employed(*Dr. John Schleifer Hoechst Roussel Vet Gillsville, GA*([www1.agric.gov.ab.ca/\\$department/deptdocs.nsf/all/pou3647?opendocument](http://www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/pou3647?opendocument)) (1).

This is a condition characterised by a sudden increase in mortality in young, typically 7-14-day-old, rapidly growing broiler chickens. Birds in good condition die after showing neurological signs. Mortality drops off as sharply as it started. This appears to be a multifactorial condition. Feed intake, and/or carbohydrate absorption are disturbed resulting in a hypoglycaemia. Males are more susceptible than females, probably because they are growing faster. Filtered intestinal contents from affected flocks appear to be capable of reproducing the condition, suggesting a viral component. In order to reproduce the typical condition the affected birds are subject to 4 hours without feed and then a mild physical stress such as spraying with cool water. (thepoultrysite.com/diseaseinfo/138/spiking-mortality-of-chickens) (2). J.B.Hess et.al reported in their study mold and mycotoxin levels in frfd from farm experiencing spiking mortality reported of all the molds identified, only *Peticillium* spp, occurred in any significant numbers.

This is not a common feed grain mold and probably doesn't present a problem in terms of mycotoxins. It is most likely a secondary growth mold occurring after pelleting. There was a low incidence of *Aspergillus spp* and *Fusarium spp* detected. Neither of these molds, the most common field and storage contaminants of grain, was present at levels expected to cause a problem in these flocks(3). The study was carried out to identify the common moulds growing in the selected feed raw materials in Owerri, Imo State, Nigeria. Fifty-four bulk samples were derived from 162 bags of 6 different raw materials, which included local fish meal (LFM), soybean meal (SBM), groundnut cake (GNC), palm kernel cake (PKC), brewers dried grain (BDG) and maize (MZ). The samples were collected during the rainy season months of June, July and August. The common moulds isolated from these samples were *Mucor spp.*, *Aspergillus spp.*, *Yeast spp.*, *Bacteria spp* and *Rhizopus spp*. More fungal organisms were isolated in the month of July although *Aspergillus spp* was not isolated during the month. Local fishmeal, palm kernel cake, and brewers dried grain had the highest isolates of three organisms each with the prevalence ranging from 13.64 to 18.18%. Soybean meal, maize and groundnut cake on the other hand returned between one and two isolates. The present result showed that untreated feed raw materials are important vehicles for introduction of fungal organisms into poultry feed. It is therefore, advised to routinely treat such feed raw materials with fungal growth inhibitors in order to limit their growth since these organisms are capable of reducing the nutritional values of finished feeds.(4) [The Journal of American Science. 2007;3(1):5-9].

Conclusion and Recommendation

As this is the primary investigation which findings indicates the mycoses related mycotoxicosis is emerging as new problem in young broiler chickens need to be evaluated to ascertain its exact cause and its suitable management need to be looked out.

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Melamine contaminated food products in Philippines

The Department of Health, Philippines on Monday, 27 Oct 2008, banned the sale and export of Sunflower Crackers Blueberry Creme Sandwich after the Philippine-made product tested positive for melamine contamination. Sunflower Crackers Blueberry Creme Sandwich is the 1st locally manufactured food product to test positive for melamine. The Hong Kong Center for Food Safety [CFS] earlier said that it found 3.2 parts per million of melamine in Sunflower Crackers Blueberry Cream Sandwich, which is above the 2.5 ppm legal limit for melamine in food products. Apart from the biscuit (cracker), the CFS said it also detected melamine in Select Fresh Brown Eggs (Extra Large, 6 piece) from China. They had a melamine level of 4.7 ppm. Hong Kong food inspectors have found eggs imported from northeast China to be contaminated with high levels of melamine, the toxic industrial additive. Scientists in China worry that in addition to adulterated dairy supplies, melamine may have been intentionally added to animal feed in China, tainting chicken and possibly fish and hog feed, which could result in poisonous meat and seafood supplies, according to a report published Sunday [26 Oct 2008] in the South China Morning Post. It accused rogue dairy producers and middlemen of intentionally spiking dairy supplies with melamine to save money, adding the chemical, which is used to produce plastics and fertilizer, as a cheap filler that can artificially inflate protein readings. **Source:** <<http://www.promedmail.org>>