Clinical Laboratory Epidemiological Investigation of hemorrhagic Proventriculitis and Gizzard Erosion in Nepal

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Abstract

A new disease condition has emerged affecting replacement pullets and broiler chickens between 2 to 10 weeks of age. The mortality rate varies between 1 to 10% and ultimately the flock is left with creation of several small poorly feathered and pale chicks. Though it has not been possible to establish the exact cause of this condition, following three points has been taken in consideration. Hemorrhagic Proliferative Proventriculitis and Gizzard Erosion is an illness that involves one or more infectious agents. One the cause suspected for it is mycotic origin. The spectrum of signs or lesions are either caused by or exacerbated by nutritional, husbandry and hygienic factors. The response to the treatment is often poor but the symptomatic treatment with toxin binder like toxolivum liquid, hepatocare, along with immunomodulaters like immunocare, and promin has significantly helped in ameliorating the mortality. Similar condition has been widely reported in broilers under various names like Malabsorption syndrome, infectious Proventriculitis, infectious runting syndrome, pale bird syndrome and stunting syndrome. Same condition is likely to be emerging as a problem for commercial broiler after 3rd week age.

Keywords: Malabsorption syndrome, infectious Proventriculitis, infectious runting syndrome, pale bird syndrome, stunting syndrome, Fungus, Penicillium, Aspergillus, Liquid Toxin binders, Broiler, chicken, Nepal.

Review of Literature

Mycotoxins T2 toxin produced by fusarium are a caustic irritant. It causes necrosis of mucosa of proventriculus, gizzard and feather epithelium. Citrinin, which is basically a nephrotoxin, can also cause fissures in the gizzard Oosporein – In oosporein poisoning one may notice that the proventriculus has enlarged circumference at the isthmus and the mucosa is covered with pseudomembranous exudates (necrosis may occur at the isthmus). Cyclopiazonic acid (CPA) – Lesions occur in proventriculus, gizzard, liver and spleen. Theproventriculus is dilated and the mucosa is thickened by hyperplasia and ulceration. Mucosal necrosis may occur in gizzard (Dr Manuel Contreras and Dr Douglas Zavieso).

Infectious Factors Adenovirus – According to case reports, gizzard erosion is characterized by adenovirus intranuclear inclusion bodies in epithelial cells. Reovirus infection could be a factor in the pathogenesis of histamine associated proventricular enlargement. High levels of Dietary biogenic amine's like histamine, 3HT, 5HT,histidine, dopamine, gizzerosine and serotonine, can be found in dietary constituents such as tankage fish meal, corn screening, soybean meal, vitamin premixes, fats, poultry meal, meat and bone meal. The biogenic amines are decarboxylation breakdown products of amino acid catabolism and these amines are considered toxic to animals. The potential for biogenic amine build-up is real in animal by product meal and is the result of breakdown of the product. Histamine is produced in the poultry feed under proper temperature and moisture conditions by microbial decarboxylation of histidine. Reduced growth, poor feathering and proventricular enlargement have been associated with histamine toxicity in chickens. Histamine toxin problem in chicken generally has been associated with the intake of fishmeal which contain high level of histidine Gizzerosine is a compound found in overheated fish meal due to interaction of casein with histidine and acts as a factor causing gizzard erosion or ulceration in chicks. It can also be formed if the temperature of fish meal increases by incorrect handling during transportation or storage particularly in hot weather (Martin D. Ficken,).

Then gizzerosine concentration of burnt fishmeal would be low because of degradation of protein.

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However it is also likely that fishmeal with good color, odor, taste and physical properties may contain a large enough quantity of gizzerosine. Gizzerosine stimulates proventricular gland secretary cells to release excessive hydrochloric acid. Gizzard lesions result from the runway digestive effects of hyperacidity. Opportunistic bacteria may subsequently colonies the nutrient rich biodetrius. The cells of the glandular alveoli of the proventriculus secrete hydrochloric acids and pepsinogen (pepsin) which is a digestive enzyme required for initial digestion of proteins. Any lesion in the preventricular glands will interfere with the secretion of pepsin with subsequent impairment of protein digestion and utilisation. The results are poor production performance, unthriftiness and poor feed conversion. This could also explain why some of the birds affected with this condition pass undigested or poorly digested feed in faeces (G. D. Butcher) .

A Reo virus strain (SS 412 stain) was isolated from an outbreak of proventriculitis/malabsorption of syndrome; the role of this reovirus strain in that outbreak was proven in experimental studies. Further studies have demonstrated that chicks from breeder hens, which have been vaccinated with oil imulsion SS 412 virus vaccine, were protected against. Proventriculitis following experimental challenge with the SS 412 reovirus strain. Anaerobic bacteria like clostridia are sometimes found as secondary invaders resulting in either ulcerative enteritis or necrotic hepatitis. It will be interesting to study the effect of viruses also since both of them cause the lesions in the proventriculus (G. D. Butcher). Other Factors Rather than ameliorating the effects of malabsorbtion syndrome, vitamin A caused a further reduction in body weight and bone ash according to a study. Supplementation of vitamin E significantly reduced both mortality and the effects of disease in body weight gain in an outbreak of pale bird syndrome in broiler chicks of 3 weeks. Amino acid imbalance (lysine and methoinine especially), excess dietary copper sulfate, lack of dietary fiber, deprivation of food and water have also been found to be responsible for the hemorrhagic proliferate proventriculitis and gizzard erosion. Lesions Proventriculi lose their normal flusiform shape and normal constriction at the junction with gizzard is diffusely enlarged and has a thickened and turgid wall. Thickening of the wall is more marked upon incising the proventriculus. The proventricular glands protrude irregularly from the mucosal surface, lose their normal pattern and contain milky fluid that could be expressed with slight pressure. The gizzard is often smaller than normal and flabby. The gizzard peels off easily with hemorrhagic ulceration of the gizzard wall (Dr. Avinash Dhawale). On laboratory analysis of sample of same grain was found to be containing 60-110CFU/gm of Penicillium spp of fungus (Karki,et.al http://www.alumbo.com/article/43263. Feed contamination can lead to nutrient losses and detrimental effects on animal health and production. Feed mould counts ranged from <1× 102 to 1× 105 cfu/g. The most frequent genus isolated was Aspergillus (40.54%), followed by Penicillium (18.38%) and Fusarium (16.22%). High levels of fungal species and mycotoxins (the toxins produced by some fungi) have been identified in equine feeds. This contamination can result in nutrient losses from the food, and negatively impact the health and productivity of horses. The most common fungi identified in this study were Aspergillus, Fusarium, and Penicillium, and the amount of fungi in the feed exceed the proposed limit of 104 cfu per gram (Stacey Oke, 2007).

Materials and Methods

Table-1. Observation of flock

Flock size	Age/week	Morbidity	Mortality
100-500 (30)	4-6	25-100	2-10
500-1000 (50)	4-6	100-200	10-20
1000-1500 (130)	4-6	200-300	20-30
1500-2000 (74)	4-6	300-350	30-35

Lesions observed during postmortem:

- Anemic appearance, poor growth, pale skin
- Mucous in URT.
- Distorted shape of proventriculus,gizzard.
- Swollen large, small intestine.
- Liver pale, enlarged with white spate.
- Enlarged spleen.
- Emphymatouse lung.
- Ascites.
- Milky fluid oozing from Proventricular gland.

Table-2. Result of Mycology Laboratory Examination of postmortem tissue samples lung,liver,spleen.

Sr. No.	Species	Total	positive	negative	Isolate fungus	No.
1	Avian	298	Fungus 182	24	Penicillium spp.	110
					Candida spp.	84
2	Avian		Bacteria spp.92		Salmonella spp	24
					Mixed E.coli and Staphylococcus spp	68

- Hemorrhage in Proventricular muscle.
- Yellow tinge with white ulceration in gizzard.

Treatment advised

Broad spectrum antibiotics, Liquid toxin binders like toxolivum toxol, Hepatocare, Immunomodulaters like immunocare, and promin for a week.

Result s and Discussion

Mycological screening of postmortem samples of liver, spleen, proventriculus of bird revealed the presence of Fungus spp like *Penicillium, Aspergillus* in 194 samples out of 284 samples were cultured in potato dextrose agar medium during the period 2007-7-1 to 2008-3-1.The postmortem lesions and age of flock had the similarities as reported by other workers. As there has not yet any observation of mycobiota of poultry lesion carried out this finding serves as preliminary information in this regard need to be further validated. As modern poultry feed milling operation toxin binder is being used but still it is not going to sufficient to combat the effect of mycotoxin produced by storage fungus. As much of the laboratory finding of this investigation ruled out the any significant involvement of any bacterial or viral agent involved in this syndrome but still histopathology of organ involved need to be done to confirm the involvement of fungus in this syndrome. This finding indicates that after 3rd week of age broiler need to be supplemented with liquid toxin binder to reduce the loss from this emerging disease syndrome need to be looked into.

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