

# Haemorrhagic Proliferative Proventriculitis And Gizzard Erosion

*A new disease condition has emerged affecting replacement pullets in India. — By Dr. Avinash Dhawale*

A new condition known as haemorrhagic proliferative proventriculitis and gizzard erosion (HPPGE) has emerged in replacement pullets in India. It affects the birds 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, most scientists and clinicians agree on following three points.

- HPPGE is an illness that involves one or more infectious agents.
- The digestive tract and endocrine organs may be primary targets for these agents.
- 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 has helped in ameliorating the mortality. According to the scientific literature similar condition has been widely reported in broilers under various names like malabsorption syndrome, infectious proventriculitis, infectious runting

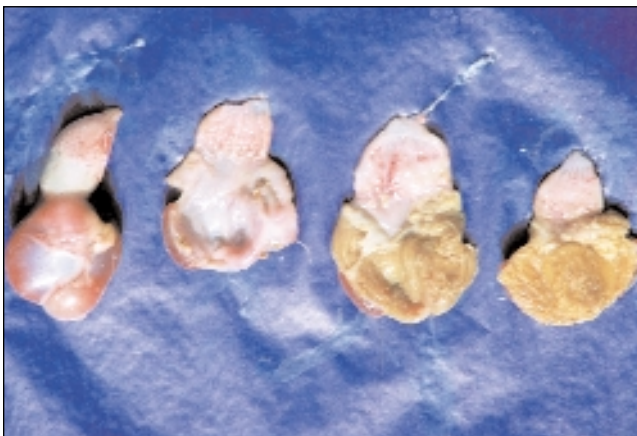
syndrome, pale bird syndrome and stunting syndrome.

## **Etiology: Non Infectious Factors -**

### **Dietary Biogenic Amines (DBA)**

High levels of DBA's like histamine, 3HT, 5HT, histidine, dopamine, gizzerosine and serotonin, can be found in dietary constituents such as tankage fish meal, corn screening, soyabean 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 fish meal which contain high level of histidine. Dietary histamine levels of 0.4 – 0.5% are required to produce constantly growth depression and other side effects. It is possible however



**Replacement pullets of 4 weeks. Loss of normal fusiform shape of the proventriculus and enlargement at the junction with the gizzard (left); Haemorrhages on the proventriculus and mild distruption of the gizzard (right).**



**Loss of normal fusiform shape of the proventriculus and enlargement of the constriction at the junction with the gizzard. (Replacement pullets - 5 weeks).**

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that the effect of ingesting lower levels of histamine may be augmented by other factors in the diet.

Gizzerosine is a compound found in overheated fish meal due to interaction of caesine 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. Then gizzerosine concentration of burnt fish meal would be low because of degradation of protein. However it is also likely that fish meal with good colour, odour, taste and physical properties may contain a large enough quantity of gizzerosine.

The pH value of the gizzard and duodenum were decreased after a week experiment in three days old chicks in a diet containing 6.25 PPM gizzerosine. The pH was 4.4 – 3.6 in the gizzard and 6.4 – 5.6 in the duodenum. This proves that gizzerosine stimulates increasing gastric acid secretion which can be inhibited by the administration of cimetidine, an antagonist of H<sub>2</sub> receptor of the stomach.

The ability of gizzerosine to introduce gastric acid secretion in chickens was almost 10 times greater than that of histamine. In addition its gizzard erosion inducing effect was 300 times greater than that of histamine. Whether gizzerosine may be formed during heating of other protein feedstuffs with high histidine content is not known. Gizzerosine is probably also produced by combustion of fish meal stacked at the bottom. The addition of 0.5 – 1% lysine hydrochloride to a diet containing fish meal alleviated the negative effect of gizzerosine on the performance of chicks.

Gizzerosine stimulates proventricular gland secretory cells to release excessive hydrochloric acid. Gizzard lesions result from the runaway digestive effects of hyperacidity. Opportunistic bacteria may subsequently colonise the nutrient rich biodebris.

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 proventricular 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.

## **Mycotoxins**

T<sub>2</sub> toxin produced by fusarium is 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 exudate (necrosis may occur at the isthmus).

Cyclopiazonic acid (CPA) – Lesions occur in proventriculus, gizzard, liver and spleen. The proventriculus is dilated and the mucosa is thickened by hyperplasia and ulceration. Mucosal necrosis may occur in gizzard.

## **Infectious Factors**

Adenovirus – According to case reports, gizzard erosion is characterised by adenovirus intranuclear inclusion bodies in epithelial cells. Reovirus infection could be a factor in the pathogenesis of histamine associated proventricular enlargement. A reovirus 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 emulsion 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.

## **Other Factors**

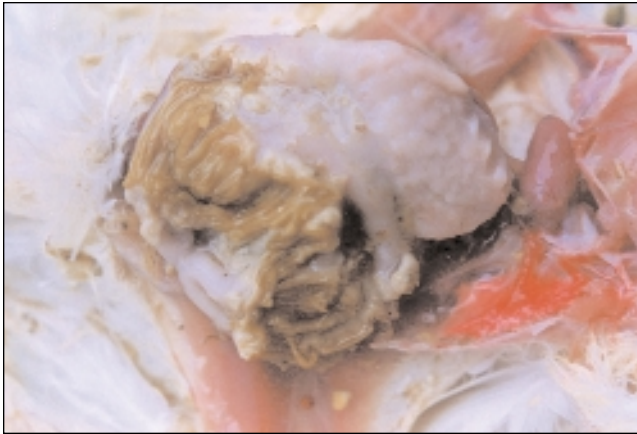
Rather than ameliorating the effects of MAS, vitamin A caused a further reduction in body weight and bone ash according to a study. Supplementation of vitamin E significantly reduced both mortality and the effects of disease in body weight gain in an outbreak of pale bird syndrome in broiler chicks of 3 weeks. Amino acid imbalance (lysine and methionine especially), excess dietary copper sulfate, lack of dietary fibre, deprivation of food and water have also been found to be responsible for the HPPGE.

## **Lesions**

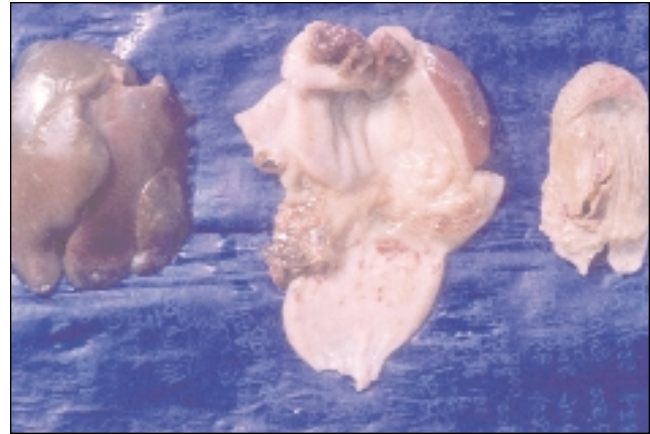
Proventriculi lose their normal fusiform shape and normal constriction at the junction with gizzard are diffusely enlarged and have a thickened and turgid wall. Thickening of the wall is more marked upon incising the proventriculus.

The proventricular glands protrude irregularly from the mucosal surface, lose their normal pattern and

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**Extensive destruction of the gizzard epithelium. (Replacement pullets – 5 weeks of age).**



**Symptoms in 9-week old replacement pullets. Showing (left) liver with white necrotic foci which in isolation it proves to be due to clostridial organisms. (Centre) Massive necrosis in the gizzard where the inner lining shows necrosis marks. (Right) Haemorrhages in the proventriculus.**

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 haemorrhagic ulceration of the gizzard wall.

## **Treatment**

- Firstly ensure optimum space per bird in the grower house. Remove excess numbers of birds if the flock is overcrowded.
- Change the litter if it is saturated.
- Stop the use of animal protein and substitute with vegetable protein sources.
- Supplementation of antitoxin preparations along with high doses of vitamin E, liver tonics, digestion stimulants, antifungals and toxin binders show remarkable recovery in the flock in 5 to 7 days.
- Addition of gut acting antibiotics to check multiplication of anaerobes like clostridial bacteria.
- We have tried to control the hyper-acidity with antacids like ranitidine, aluminium hydroxide etc. through water: The result is quite encouraging.
- It is necessary to correct the dietary amino acid balance, crude fibre level, calcium and phosphorus etc.
- Addition of enzymes to the feed since pancreatic activity also seems to be adversely affected in HPPGE.
- It is essential to establish the involvement of reo- or adenoviruses and do the necessary amendments in the breeder vaccination schedule. — *Dr. Avinash Dhawale Nagpur, India.*