Gout & High Mortality in Indian Broiler

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Abstract – This paper is about the new approach to control early chick mortality due to gout. Indian Broiler farmers are suffering from high mortality during every monsoon which starts as early as 5 days age with kidney swelling & Gout. Mortality is between 5-10% if controlled by proper management, otherwise mortality continues till marketing & ends even up to 25%. Considering the history, epidemiology, clinical signs, post mortem findings & response to different control measures, it has been suggested primarily as Inclusion Body Hepatitis (IBH) predisposed by subclinical mycotoxins. This IBH in most cases been followed by Infectious Bursal Disease (IBD) and finally Chronic Respiratory Disease (CRD) resulting these high mortality.

Keywords – Gout, Epidemiology, IBH, Mycotoxins, IBD, CRD

1. *Epidemiology*

Due to the presence of chalky white depositions in the kidneys and the surface of visceral organs including heart, most people call it as gout and handling the issue very casually.

Gout or Avian Urolithiasis is not a single disease entity, rather the result of kidney damage from a number of potential causes, like Infectious disease, Nutritional issues, toxic substances, or a combination of such factors [1].

During monsoon, this situation arrives in most part of India, when moisture content of feed ingredient like maize, GNC, etc as well as finished feed increases. This high moisture content results in mould growth in finished feed which may not cause clinical Mycotoxicosis all the times. But combined effect of several sub-clinical levels of mycotoxins regularly causes immunosuppression. Immunosuppression at early stage of chick's life resulting easy entry of infections like IBH, IBD, followed by *E coli*, CRD, etc at later stage.

Both IBD & IBH are immunosuppressive in nature and invites each other in field condition resulting high mortality in chicks. Immunosuppressed chicks never grow well and secondary infection like *E coli* & CRD is eminent resulting consistent slow mortality with poor productivity.

2. *Pathology*

Uric acid, the end product of protein metabolism in birds excretes through kidneys. Damage to kidneys due to any reason disturb normal renal function leading to failure of kidneys to excrete the uric acid leads to high Uric acid level in blood (hyperuricaemia) resulting deposition of monosodium urate crystals in the kidneys as shown in Fig.2 and mesentery, peritoneum, heart, liver, air sacs, joints, etc as shown in Fig.1.

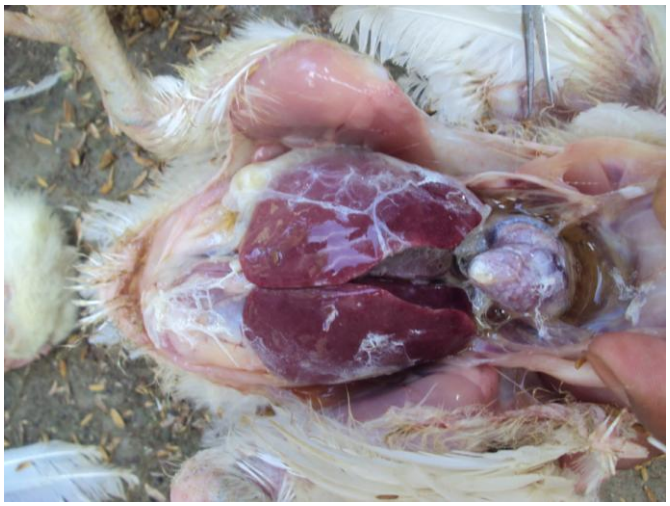

Fig.1. Chalky Urates on Visceral organs

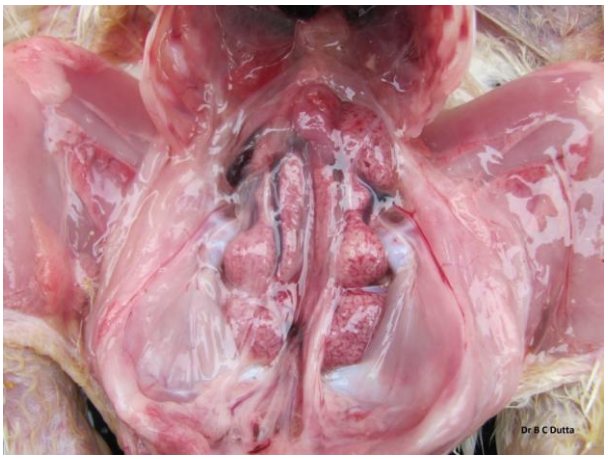

Fig.2. Urate deposition in Kidneys

The damaged kidneys are characterized by atrophied or missing portions of kidney lobes, kidney & ureters stones and remaining kidney tissues become swollen & white with full of urates. Compensatory enlargement of remaining normal kidney tissue takes place to maintain

adequate renal function. Birds usually die from renal failure.

3. Causes

The cause of gout is often difficult to determine. The actual kidney damage may occur long before the onset of mortality. The possible factors that contribute to gout are Nutritional, Infectious, toxic or any combination of these.

3.1 Nutritional

3.1.1 Excess dietary calcium over a period of time.

3.1.2 Low Phosphorus (act as urinary acidifier) content in feed.

3.1.3 Excess Sodium through excess Salt or Sodium bicarbonate in feed or in drinking water.

3.1.4 Vitamin A deficiency over a long period of time.

3.1.5 Water deprivation – mechanical malfunction of kidneys. Dehydration of hatching eggs due to poor storage & transportation and dehydration of newly hatched chicks due to poor hatchery management, excess holding time, poor transport system & poor brooding management

3.1.6 Very excess protein intake – increased uric acid production may lead to gout.

3.2 Infectious

3.2.1 Infectious Bronchitis (IB) – affect respiratory tracts along with kidneys. Abnormal respiratory sounds, gasping, sneezing, nasal discharge, facial swelling, pale swollen kidneys with urate deposits, high mortality up to 25% [2]. On post mortem yellow cheese-like exudates in air sacs are common and caseous plug may be found in severe case.

3.2.2 Avian Nephritis virus – pale & swollen kidneys with urate deposits are common. Day old chicks mostly affected.

3.2.3 Inclusion Body Hepatitis (IBH) caused by Adenovirus – kidney swelling with urate deposits occurs as early as 5-6 days age. Mortality reaches its peak on 8th or 9th day with pale yellowish liver & white kidney as shown in Fig. 3 & Fig. 4. The disease can be differentiated from other similar conditions with hydropericardium, sometime with blood as shown in Fig. 5 and atrophy of Immune organs like bursa, thymus & spleen.

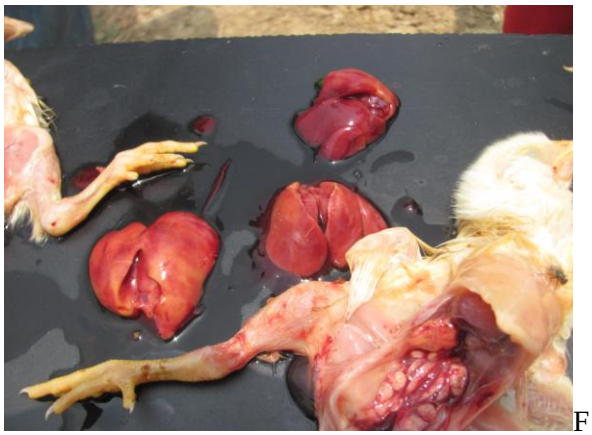

Fig.3. Pale enlarged Liver & urates in kidney.

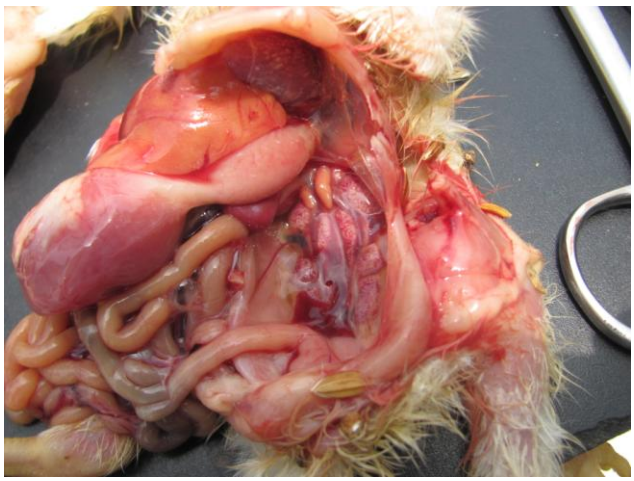

Fig.4. Enlarged Yellow Liver

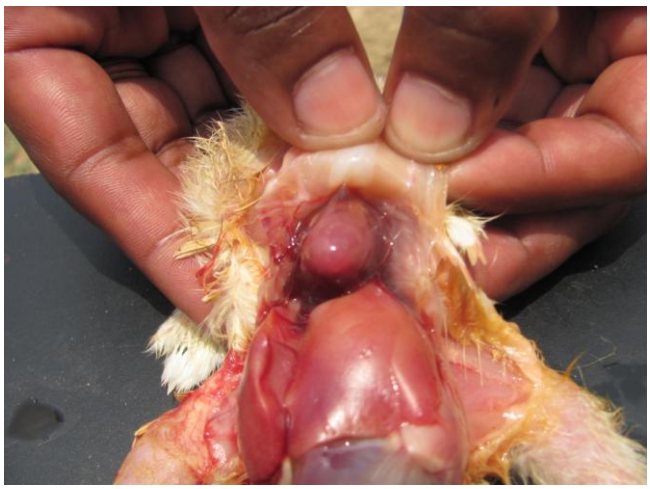

Fig.5. Hydropericardium

3.2.4 Infectious Bursal Disease (IBD) – Sub-clinical IBD in early age causes swelling of kidneys with urate depositions along with pathognomic changes in Bursa [3], which may vary in size with sticky to cheesy secretions inside as shown in Fig. 6 & Fig.7.

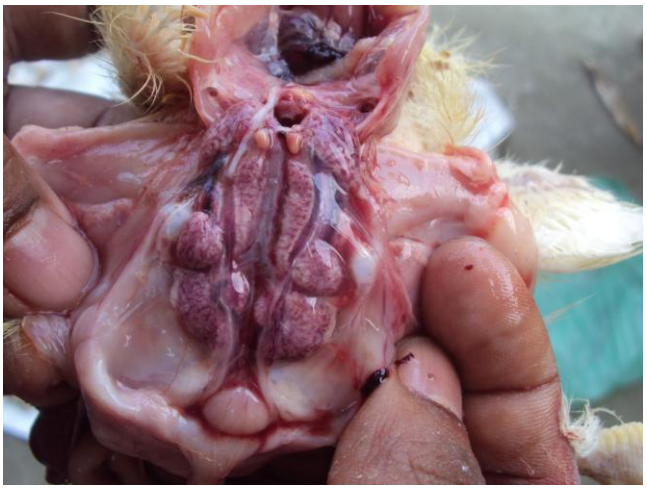

Fig.6 Congested Kidneys with urate deposits

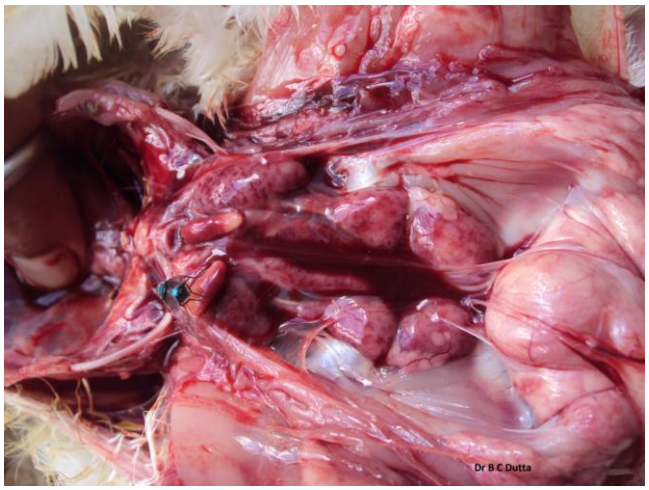

Fig.7. Enlarged Bursa with Kidney Urates

3.3 Toxins

3.3.1. Antibacterial, such as Sulfas and aminoglycosides like Gentamycin, Neomycin, etc are nephrotoxic.

3.3.2. Disinfectants & Insecticides (e g Ethylene dibromide) – any dose miscalculation

3.3.3. Mycotoxins (fungal toxins) like ochratoxins (in maize, rice gluten, etc), aflatoxins & oosporein (in maize, rice gluten, GNC, etc), dioxynivalenol or DON (in maize, rice gluten, wheat, etc) cause kidney damage resulting gout [4]-[6].

4. Control

Gout is a multi-factorial problem and identifying a specific cause is often difficult. History and field cases have demonstrated the importance of interactions between 2 or more contributing factors especially Mycotoxins & virus infections.

The condition occurs every year with the onset of monsoon and with the arrival new harvest Maize. The high moisture contents of Maize (>15%) and finished feed (>12%) favors the growth of moulds specially when stored for long. It has been seen that these Gout problem is more when the time gap between feed production & consumption is longer.

The Laboratory test of maize sample & finished feed in almost all cases confirms presence of various toxins in clinical or subclinical level. Even when individual toxins are below to toxic level, combined Mycotoxicosis (combination of Aflatoxins, Ochratoxins, DON, and oosporein) can cause the damage. Individual Mycotoxins even @ 5ppm may not cause clinical Mycotoxicosis but may cause immune-suppression leading to easy entry for viruses resulting diseases.

Both IBH & IBD are immune-suppressive disease and one can predispose other aggravating the situation.

During monsoon with fungus infested feed, the chicks are getting toxins even before the maternally derived antibodies developed immunity in the body. Immuno-suppression results following intake of such feed.

IBH viruses, present in the poultry environment, which normally cannot infect healthy chicks, invade the immune-suppressed chicks and the disease develops within 5-7 days age. Chicks look apparently healthy with normal feed consumption but symptoms of ruffled feather can be observed. Chicks die without showing any symptoms.

Poultry environment is loaded with IBD virus because of the resistant nature of the virus. Immune-suppressed chicks easily gets the IBD infections as early as 5-6 days age and the signs of disease can be observed from 8 days onwards with lesions in Immune organs like Bursa, Thymus & Spleen which shows degenerating changes with varying sizes. Kidneys face more damage by both IBH & IBD virus resulting heavy gout mortality.

Considering the history, etiology & pathogenecity, the following measures may helps controlling mortality:

4.1. Biosecurity:

4.1.1 Shed Ready – Inside & outside of the poultry shed shall be made disease free by proper cleaning, washing, disinfections before housing of chicks.

4.1.2 No Entry of Infections – Complete fencing with well maintained Foot-Bath at entry points is mandatory. Visitors absolute no entry. Production staff shall enter only

after following biosecurity norms. Production supervisors shall not enter inside the shed.

Fig. 8 Bio-security (Fencing with Foot bath)

4.1.3 Do not spread Infections – Disposal of mortality by burial or in death-pit with formalin inside the fenced area is mandatory. Supervisor shall not visit any other farm after visiting infected farm.

Fig 9. Bio-security (Death pit)

4.1.4 Use of Disinfectants & Sanitizer to reduce existing infection load –

4.1.4.1 Regular spray of potent disinfectant in presence of birds inside & outside of the shed.

4.1.4.2 Use of good Water sanitizer from day one to culling.

4.2. Avoid Stress to Chicks due to

4.2.1 Poor Hatchery Management – Issues are ventilation, sanitation, humidity, hatch window, Temperature, etc.

4.2.2 Long Chicks holding time – need to house chicks as soon as possible after pull-out.

4.2.3 Poor Transportation system – issues are time, duration, ventilation in transit, etc

4.2.4 Faulty Brooding – issues are Temperature, Ventilation, Drinking water, space, draft, etc.

4.2.5 Over-medication or over use of salts or Sodi bi-carb.

4.3. Feeding Management:

4.3.1 Manufacturing point –

4.3.1.1 Use of Ingredients with accepted level of Moisture.

4.3.1.2 Use of good Toxin binder & Mould Inhibitor

4.3.1.3 Use of good Acidifier combination.

4.3.1.4 Moisture control in finished feed.

4.3.2 Transportation & Storage at godown or at farm–

4.3.2.1 Minimize transportation time

4.3.2.2 Protect from seepage of rain water on to feed bags

4.3.2.3 Minimize storage time with the objective of providing fresh feed to chicks. In monsoon with high humidity & high moisture in feed, all feed must be consumed within 5-7 days of manufacturing. Therefore, godown shall store feed for 1 day only. Farm shall never store feed more than 3 days.

4.3.3 Feeding practice at farm -

4.3.3.1 Feed bags shall be kept on Wooden platform leaving space from walls

4.3.3.2 Keep space between 2 rows of feed

4.3.3.3 Frequent Feeding, at least 3 times a day

4.3.3.4 Emptying of Feeder once daily and cleaning of feeder base, bucket & cone daily.

4.4. Vaccination & Medication:

4.4.1 Schedule & selection of Vaccine Strain shall consider the following –

4.4.1.1 Disease history of the area & the farm itself

4.4.1.2 Maternal Antibody level of the day old chicks

4.4.1.3 Vaccination schedule shall aim at controlling sub-clinical IBD, may be by booster vaccination after priming.

4.4.1.4 Medicine schedule must consider the factors to improve the immunity; by using good Probiotics from very 1st day. Use of Vit E with Selenium is very effective

4.4.1.5 Unnecessary use of Antibiotics shall be avoided as much as possible. Nephrotoxic antibiotic items like Gentamycin, Sulfas should be avoided.

4.4.1.6 Use of Acidifier in drinking water is very effective in many ways – reduces pH, neutralizes toxins of feed; lower pH prevents growth of E coli & other harmful bacteria and stimulates growth of helpful bacteria like *Lactobacillus*.

4.4.1.7 Proven disinfectant shall be sprayed before 24 hours of chick housing to make the brooding house free of microbes.

4.4.1.8 Good disinfectants shall be spread regularly inside the poultry shed in presence of birds to prevent horizontal spread of infection.

4.4.1.9 Use of Potassium Chloride or Jaggery water is effective in controlling gout mortality.

5. *Conclusion*

Poultry needs a professional approach to sustain in current scenario when many harmful viruses are present in poultry environment. Steps shall be taken to select the right feed ingredients, that too free from moulds. Because Mycotoxins are the silent killer and the prime factor of inviting the harmful viruses in today's poultry. Steps shall be taken to control even low level of mycotoxin in feed.

Biosecurity is the other area to be taken care. Implementation of comprehensive biosecurity practice needs a change in approach of conventional poultry farming. All breeding farms should be a restricted area for all and commercial farms shall plan to upgrade his biosecurity practice to the standard of a breeding farm. The 3 chapter of biosecurity needs to be implemented in all poultry premises in order to prevent this mortality are; prevent entry of infections in poultry premises, minimize existing infection loads and do not spread infections.

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