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Short Communication

INCIDENCE AND CLINICO-PATHOLOGICAL STUDY OF GOUT IN BROILER BIRDS OF FAIZABAD AND SULTANPUR DISTRICTS OF EASTERN UTTAR PRADESH

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ABSTRACT: The study was conducted in 12 different private and commercial broiler flocks for one year. Out of 23906 broiler birds examined, 1714 were clinically found to be naturally affected with gout, the incidence and mortality of gout being 7.17% and 4.37% respectively. The highest prevalence of this disease was recorded during colder months (833, 9.68%, 439, 7.06%) followed by summer (384, 5.40%) and lowest in rainy season (58, 2.92%). Of the susceptible age of first three weeks, the incidence was more during second week (9.96%), followed by first (7.63%) and third week (6.01%) of age. Out of the 12 flocks which were screened for gout 10 flocks were of cob breed (83.34%), one flock of Hubbard (8.33%) and one of Kasila breed (8.33%). The birds affected with gout had a significant (P=0.01) increase in serum uric acid (34.42 ± 1.10). The levels of serum creatinine were significantly (P>0.01) increased (2.14 ± 0.02). The mean values of TLC and PCV in the affected birds were significantly (P>0.01) high. TEC and Hb were also significantly (P>0.05) higher than control birds.

Key words: Broiler birds, Gout, Incidence, Pathology.

Gout is a condition in which high level of uric acid in the blood (hyperuricaemia) lead to deposition of urates on the surfaces of various internal organs or various joints especially the hock joint and responsible for a great deal of morbidity (sickness) and mortality (deaths) in both broilers and layers. Gout is more a sign of renal damage than a distinct disease (Sathiyaseelan *et al.* 2018, Namratha *et al.* 2019). Some managemental stress factors including high brooding temperature and higher level of ammonia concentration in the shed can also cause gout (Bulbule *et al*. 2014). Uric acid is the primary catabolic product of protein, non-protein nitrogen and purines in birds. It is synthesized mainly in the liver of birds. Birds excrete uric acid as primary nitrogen metabolite as calcium and sodium urates which are not water soluble. This is called uricotellic mechanism (Pathak 2002). But any injury or damage to bird's kidney, from whatever cause, interferes with the elimination of uric acid, which then accumulates in the blood (hyperuricaemia) and leads to gout *i.e.*, deposition of urate crystals in various tissues (Jordan 1990). Visceral gout assumed prime importance than articular gout as it causes regular mortality, leading to great economic losses to poultry industry. Keeping in view the above facts, the present study was conducted to record the incidence of gout in broiler birds in organized and unorganized farms in Sultanpur and Faizabad districts of Eastern Uttar Pradesh. and to record the haemato-biochemical changes in gout affected birds.

Incidence and haemato-biochemical study

For this study, broiler birds affected with gout were screened on the basis of clinical signs viz. loss of body weight, reduced feed intake, dull appearance and by observing specific chalky white deposits on visceral organs during postmortem examination of dead birds in 12 different private and commercial broiler flocks located in Faizabad and Sultanpur districts of Eastern Uttar Pradesh for one year starting from June 2015 to May 2016. The private farms were selected on the basis of stratified random sampling. The chalky white deposits from visceral organs muscles, joints were collected by scraping with a blunt knife for identification of uric acid crystals by microscopical examination and murexide test mentioned by Sharma (1977).

The incidence of the disease was studied in relation to age groups and mortality pattern of affected birds. Seasonal

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variations of the disease were recorded for one year, dividing the whole year in four quarters viz. 1st (July to September), 2nd(October to December), 3rd (January to March) and 4th (April to June).The nature and clinical manifestation of the disease were thoroughly studied from the onset of the disease. For the study of gross pathological lesions, detailed examination of all carcasses was performed meticulously soon after death.

For Serum biochemical study, 5.0 ml of blood was collected from moribund birds showing clinical signs as well as from normal healthy birds to serve as control. Serum uric acid (mg/dl) was estimated photometrically by modified carbonate phosphotungstate method (Henry *et al*. 1957).The serum creatinine (mg/dl) was estimated photometrically by Alkaline Picrate method (Bonsnes and Taussky 1945).Serum phosphorous (mg/dl) was estimated photometrically by Ammonium Molybdate Method (Wang *et al*. 1983).Serum calcium concentrations (mg/dl) were determined in AAS as per the method described in the instrument manual "Analysis of serum and plasma: Calcium and Magnesium", Atomic Absorption Spectroscopy, Perkin-Elmer Corporation.

For haematological study, blood was collected from wing vein of the affected birds with sterilized needles and syringe. Ethylene diamine tetra-acetic acid (EDTA@1mg/ml) was used as anticoagulant. Haemoglobin (Hb) level, Packed Cell Volume (PCV), Total erythrocyte count (TEC), Total Leucocyte count (TLC) and Differential Leucocyte count (DLC) was estimated as per Chauhan and Roy (1998) with minor modifications.

Out of total of 23906 broiler birds examined, 1714 were clinically found to be naturally affected with gout. The overall incidence of the gout being 7.17% which corroborated with the findings of Srivastava *et al.* (2002) and Jana *et al*. (2008) who also reported the incidence of the disease in broiler birds ranging from 6.25 to 12.86% and 7.03% respectively. The results indicated that gout was very common among broiler birds in Faizabad and Sultanpur districts of Uttar Pradesh. There exist other reports indicating the incidence of the disease by Uma *et al*. (1996), Talha *et al*. (2001) as 1.69%, 0.79% broiler birds respectively, which were in contrast with present findings. Such difference could be ascribed to variations in nutritional status, husbandry practices from region to region and even within the farms of the same place. In the present study, the overall mortality being 4.37% simulated the findings of Srivastava *et al*. (2002) and Jana *et al*. (2008) who reported mortality ranging from 1.0 to 5.5% and 4.67% respectively but was in contrast with the findings of Miksch *et al.* (2002). This was probably due to variation of geoclimatic conditions, nutritional, toxic and patho-physiological conditions.

The results on seasonal incidence of the disease showed its higher prevalence during colder months *i.e.* October to March, followed by summer (April to June) and lowest in rainy season (July to September). Srivastava *et al*. (2002) and Jana *et al*. (2008) also observed higher incidence during colder months. Higher mortality rate during colder months might be due to cold stress. Such difference might be due to variation in temperature, relative humidity and husbandry practices as well as presence of infectious agents (Chowdary 1988, Sathiyaseelan *et al*. 2018). Higher prevalence of gout in rainy season than summer season could be ascribed to poor hygienic conditions prevailing during rainy season.

Of the susceptible age of the first three weeks, the incidence was more during second week (9.96%), followed by first week (7.63%) and third week (6.01%) of age. The present observation corroborated with the observation of Jana *et al.* (2008). But partially with that of Srivastava *et al.* (2002) who also recorded the more incidences during second week of age. Out of the 12 flocks which screened for gout, 10 flocks were of cob breed (83.34%), 1 flock of Hubbard (8.33%) and one of Kasila breed (8.33%).

In the present study, it was observed that the onset of the disease was sudden. Clinically, the spontaneously affected birds showed no specific clinical signs as also reported by Chowdary (1988). The birds showed dull appearance,

Fig.1. Chalky white urate deposition on enlarged liver and heart of gout affected birds.

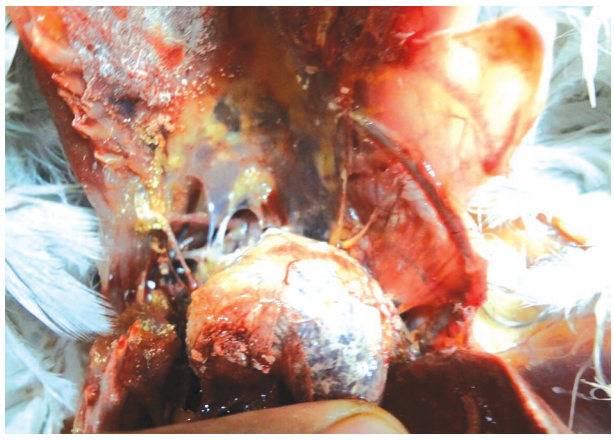

Fig.2. Urate deposition on the surface of heart and breast muscle of gouty birds.

Table 1. Haemato-biochemical values in gout affected and control birds (mean value ± SE).

Parameters	Control (10)	Affected (20)
Uric acid (mg/dl)	5.58 ± 0.14	34.42 ± 1.10**
Creatinine (mg/dl)	0.89± 0.01	2.14 ± 0.02**
Calcium (mg/dl)	9.16 ± 0.07	8.89 ± 0.08
Phosphorus (mg/dl)	5.83 ± 0.08	5.46 ± 0.07
Hb (gm/dl)	9.12 ± 0.14	10.14 ± 0.15*
PCV	29.47± 0.29	38.13 ± 0.47‘5**
TEC (10^6/Cumm)	3.48 ± 0.12	4.26 ± 0.08*
TLC (10^3/ Cumm)	22.36 ± 0.48	26.89 ± 0.41**
Heterophils	34.53 ± 0.51	28.53 ± 0.56**
Lymphocytes	52.20 ± 0.47	62.87 ± 2.99**
Eosinophils	5.33 ± 0.32	3.53 ± 0.41**
Basophils	1.40 ± 0.13	0.40 ± 0.13*
Monocytes	6.53 ± 0.26	4.67 ± 0.21

** P < 0.01

Numbers in the parenthesis indicate number of birds.

unthriftiness, reduced feed intake resulting in uneven sizes, gradual emaciation, loss of body weight, polyurea and diarrhoea. Altman *et al*. (1997), Jana *et al*. (2008), Sathiyaseelan *et al.* (2018) and Namratha *et al.* (2019) also described the similar clinical signs.

In the present study, white chalky deposits were collected from visceral organs, muscles, joints of 75 dead gouty birds to perform murexide test. In all cases, this test gave positive clinical diagnosis by the occurrence of uric acid crystals in the chalky white deposits. Rao *et al.* (1993) and Jana *et al.* (2008) also performed this test for clinical diagnosis.

The biochemical profile of both affected and control birds are presented in Table 1 which showed that the birds affected with gout had a significant (P<0.01) increase in serum uric acid (33.85 ± 1.09). Presumable, as suggested by Chandra *et al.* (1985), this elevation in the level of serum uric acid might be due to failure of excretory function of kidney and to the increased degradation of nucleic acid released as a result of degenerative changes in various organs. Mishra *et al.* (1981) stated dehydration might be responsible for slight increase in uric acid level. Results also showed that the levels of serum creatinine were significantly (P<0.01) increased (1.96 ± 0.02). These findings corroborated with the findings of Jana *et al.*(2008).There were no significant changes in serum calcium and phosphorus of gout affected birds compared to control birds. Cosgrove (1962) also reported inorganic phosphorus in affected birds appeared normal. It could be due to starvation, insufficient absorption, body fluid imbalance as well as the low level of Ca^{++} was due to increased metabolic activity of the degenerating organs. Namratha *et al.* (2019). Li *et al.* (1998) and Namratha *et al.* (2019) reported increased calcium level which was due to intake of high Ca diet.

Systemic response of gout in broilers was reflected by changes in haemograms of the affected birds. The mean values of haematological profiles of both control and affected birds are summarized in Table 1. The mean values of Haemoglobin (Hb) and Packed Cell Volume (PCV) of the affected birds were significantly (P<0.05, 0.01) higher than control. Total Erythrocyte Count (TEC) of the affected birds were significantly (P<0.05) higher than control. The result showed that the mean Total Leukocyte Count (TLC) of the affected birds were also significantly (P<0.01) higher. The result in present study corroborated the findings of Chandra *et al.* (1985). The observation of increased levels of PCV, TEC, Hb values were possibly due to dehydration as a result of diarrhoea (Chandra *et al.* 1985). These findings confirmed the earlier report of Jana *et al.* (2008). The leucocytosis in the affected birds was mainly contributed by the absolute increase of lymphocytes was suggestive of the onset of cellular immunological response of the body of affected birds to any infection.

Gross pathological lesions

Rapid screening examination of all the birds disclosed dehydrated carcasses. Chalky white deposits were seen over the subcutaneous tissues, liver (Fig. 1), kidney, heart (Fig. 1, 2), lungs, spleen, surface of the breast muscle (Fig. 2) and serosal surface of the gastrointestinal tract and in air sacs.

The pericardium was thickened and had a plaster like appearance (Fig. 2). The kidneys were enlarged with urate deposition in ureters, harder in consistency. Liver was enlarged, friable and congested. Lungs were edematous, congested. These gross lesions recorded in the present study, confirmed the earlier reports of Eldaghayes *et al.* (2010), Feizi *et al.* (2012) and Namratha *et al.* (2019).

In some cases, along with surface of visceral organs, articular surface particularly hock joints, revealed white chalky urate deposition. The joints were enlarged and swollen. On opening the joints, white semifluid deposits of urates were found within the joints. So, visceral and articular gout occurred concurrently in some cases which simulated the reports of Calnek *et al.* (1994), Jana *et al.* (2008) and Namratha *et al.* (2019). Urate deposition was generally due to failure of urinary excretion. This might be due to obstruction of ureters, renal damage or dehydration.

Gout is a baffling problem in broiler industry as it is responsible for a great deal of morbidity and mortality in broiler birds. The incidence of gout is more during first weeks of age. The highest prevalence of this disease was recorded during colder months. But from history, clinical signs, pathological and haemato-biochemical studies, it

might be concluded that kidney damage is surely to occur in case of gout.

REFERENCES

Altman RB, Clubb SL, Dorrestein GM and Quesenberry K (1997) Avian Medicine and survey. W.B. Saunders Company. pp 632-633.

Bulbule NR, Kapgate SS, Chawak MM (2014) Infectious causes of gout in chickens. Adv Anim Vet Sci 2(4): 255-260.

Bonsnes RW and Taussky HH (1945) On the calorimetric determination of creatinine by the Jaffe reaction. Journal of Biological Chemistry 158: 581.

Calnek BW, Barnes HJ, Beard CW, Reed WM and Yoder HW (1994) Disease of poultry. 9th edn. pp 732. New Delhi, Affiliated East West Press Pvt. India.

Chandra M, Singh B, Gupta PP, Ahuja SP and Singh N (1985) Clinicopathological, haematological and biochemical studies in some outbreaks of nephritis in poultry. Avian Diseases 29: 590-600.

Chauhan HVS and Roy S (1998) Poultry Disease Diagnosis and Treatment. 2nd edn. New Age International (P) Ltd., Publisher New Delhi.

Chowdary C (1988) Visceral gout in poultry. Poultry Guide XXV (9): 23-32. Cosgrove AS (1962) An apparently new disease of chickens –Avian Nephrosis. Avian Diseases 6: 385-389.

Cosgrove AS (1962) An apparently new disease of chicken - Avian nephrosis. Avian Disease 6: 385-389.

Eldaghayes IM, Hamid MA, El-Attar SR and Kammon AM (2010) Pathology of Gout in growing layers attributed to high calcium and protein diet. Int. Sci. Res. J 2 (4): 297-302

Feizi A, Haghigat A and Taifebagerlu J (2012) Assesment of Clinical and Necropsy Manifestation, Losses and Some Biochemical Factors in Broiler Breeder with Gout Syndrome. J. Ani. Vet. Adv 11(20): 3705-3708.

Jana S, Niyogi D, Mandal S, Ganguly S and Mukhopadhaya SK (2008) Haematological and Serum biochemical alteration in gout of broiler birds. Indian J Anim Hlth 47 (2): 113-115.

Jordan, F.T.W. (1990). Poultry Disease. 3ed edn. University Press, Cambridge.

Li XM, Deng MX, Li QY, Jia RY and Wang J (1998). Experimental study on the clinical pathology of urate deposition in chickens. J Vet Sci 18 (1): 49-51.

Henry RG, Sobel S and Kim J (1957) A modified carbonate phosphotungstate method for the determined of uric acid. Am J Clin Pathol 28: 1-6.

Miksch K, Grossman E, Hohler K and Johne R (2002) Detection of Goose Haemorrhagic Polyoma Virus (GHPV) in flocks with haemorrhagic nephritis and enteritis of geese in Southern Germany. Berliner-und-Munchener-Tierartliche-Wochenschrift 115 (9-10): 390-394.

Mishra SK, Sharma UK and Singh NP (1981) Biochemical observation in poultry fed diets containing sodium bicarbonate. Indian Journal of Poultry Science 16: 201-205.

Namratha L, Kumar YR and Lakshman M (2019) Pathology of visceral gout in layer chicken. Int J Recent Sci 10 (10): *35546-35548.*

Phatak RK (2002) Management of gout in tropical countries. Poultry Forture. *March Volume,* pp 31-33.

Rao TB, Das JH and Sharma DR (1993) An outbreak of gout in poultry in East Godavari district. Andhra Pradesh. Poultry Advisor XXVI (IV): 43-48.

Sathiyaseelan M, Kumar R, Lakkawar AW, Nair MG, Thanislass J and Varshney KC. (2018) Pathology of Gout in Commercial Broiler Chicken. Int. J. Curr. Microbiol. App. Sci 7(7): 2414-2417.

Sharma GL (1977) Miscellaneous Pathological Conditions and Disease in Hand Book of Animal Husbandary. ICAR Publication, New Delhi-410.

Srivastava N, Joshi BP, Jani PB and Prajapati KS (2002) Epidemiological studies on visceral gout in broiler chicks. National symposium on Recent Advance in Diagnosis of: Livestock, Poultry and Wildlife with special Emphasis on Hill Area Diseases and XIX Annual Conference of Indianb Association of Veterinary Pathologists. Palampur, Hamnchal Pradesh, India.

Talha AFSM, Hossain MM, Chowdhury EH, Bari ASM, Islam MR and Das PM (2001) Poultry disease occurring in Mymen Singh district of Bangladesh. Bangladesh Veterinariium 18 (1): 20-23.

Uma CA, Vijayasarathi SK, Sreenivasa GRN and Sathyanarayana ML (1996) Incidence of gout in poultry. Indian Journal of Poultry Science 32 (3): 237-238.

Wang J, Chen CC and Osaki S (1983) Optimisation of the phosphorous-UV reagent. Clin Chem 21: 1255.

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