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Establishment of gout induction model through sodium bicarbonate intoxication in broilers

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Abstract
The gout condition is regularly occurred in commercial poultry farming in spite of various treatment options. Hence, it's highly essential to formulate a suitable remedy for gout irrespective of etiology and to study the antigout activity it's a prerequisite to standardize a suitable gout induction model in broiler chicken. In this context, the present study was conducted in forty broiler chicks of 10 days old divided into five groups with triplicate as per experimental design. The gout was induced by dissolving toxic dose of sodium bicarbonate (SBC) powder in drinking water at different rates (5 g/ L. 10 g/L, 15 g/L and 20 g/L) on 11th day of age. The blood samples were collected on day 10 (before induction), 15 and 18 for serum uric acid and creatinine estimation. The clinical manifestations, mortality due to gout induction were noted and gross and histopathology studies were conducted. The data collected on biochemical parameters were analyzed statistically by One-way ANOVA using SPSS software (version 20). The gout signs like watery droppings, dullness, depression and unthriftiness and mortality were occurred within three days in 20 g/L group and higher serum uric acid (24.13±0.97 mg/dL), creatinine (0.73±0.03 mg/dL) values were noted on day 15. Further, gross pathology and histopathology findings confirmed development of gout in broiler chicken. Hence, it was concluded that administration of SBC @ 20 g/L could be a reliable model to study the antigout activity of any medicinal preparations in broiler chicken.

Keywords: Gout induction, sodium bicarbonate, uric acid, broiler chicken

1. Introduction
Gout is a common metabolic disorder which results in abnormal accumulation of urates (Damodaran *et al*., 1978) [3] since, the uric acid is the end product of nitrogen metabolism (Riddell *et al*., 1997) [15] in birds. The occurrence of gout condition results in severe economic losses to the farming community. The identification of the etiological agent in field cases is difficult since one or more factors can contribute to the development of gout in birds *viz*. infectious agents, nutritional causes and managemental factors. Inspite of different etiological factors the toxicopathological changes are virtually similar in broiler chicken. Although various allopathic or ayurvedic combinations were tried in the field conditions but the treatment of gout is very much challenging. Hence in order to develop an antigout formulation the prerequisite is to establish a suitable model resembling gout condition.
Excess sodium bicarbonate is one of the causative factor results in gout in birds. Generally, the addition of sodium bicarbonate to the poultry feed improves body gain and decreases losses due to heat stress and useful to correct disorders of acid base balance. Further the laxative effect of sodium bicarbonate was documented when it was added to drinking water and its addition to ration increases the egg shell thickness in layers. The aforementioned beneficial effects of sodium bicarbonate can be only attained when the recommended doses are administered. Toxicity due to excess sodium bicarbonate administration has been reported and nephrotoxic effect of sodium bicarbonate overdosing was documented by Davison and Wideman (1992) [4]. Hence in the present study has been formulated to identify the suitable toxic dose of sodium bicarbonate which causes gout in birds and to find out the toxicopathological changes due to gout induction and to establish an appropriate model to

induce gout in broiler chicken in order to study the effect of allopathy or ayurveda preparations in future.

Materials and Methods

The *in vivo* studies were conducted after obtaining the approval from the ethical committee. The day old broiler chicks were reared in caged houses under standard and uniform managemental conditions. The gout induction study was carried out by administration of sodium bicarbonate in drinking water to broiler chicken as per Mubarak and Sharkawy (1999) [13] and Sodhi *et al.* (2008) [17]. Forty day old broiler chicks were procured and maintained under standard management practices for eighteen days. They were divided into five groups of eight each with duplicate as per the experimental design shown in table 1. The gout was induced by sodium bicarbonate dissolved in drinking water at the age of 11th day in all groups, except control. The manifestation of symptoms from the day of gout induction was observed in all treatment groups. Occurrence of mortality was promptly monitored in all treatment groups. At the instance of mortality, the post mortem was conducted to record the gross pathological lesions. Simultaneously, blood samples were collected on fourth day after gout induction (day 15) for serum uric acid and creatinine analysis. Based on the results of gross pathology and serum biochemical parameters, sodium bicarbonate administration in drinking water was discontinued in T_5 group after four days whereas other group of birds received their respective dosing for one week. Blood samples were collected again on seventh day after gout induction (day 18) for serum uric acid and creatinine analysis and on same day all birds were slaughtered and gross and histopathology examination (Bancroft and Gamble, 2008) [1] was conducted. Also special stain (Von Kossa) was used to examine the urate crystals deposition in kidney of gout induced broiler chicken.

Complete randomized design was followed for the experiments (Snedecor and Cochran, 2004) and the data collected on various parameters were analyzed statistically by One-way ANOVA using SPSS software (version 20). Post-hoc analysis was done by Duncan's significance difference test. Results are expressed as mean ± SE.

Results

Gout induction study was conducted in broiler chicken by oral administration of sodium bicarbonate (SBC) dissolved in drinking water at various concentrations from 11th day to 14th day of age to fix the effective concentration for gout induction in broiler chicken. The effective dose was determined using the values of serum uric acid, serum creatinine, clinical signs and mortality rate due to gout induction and gross pathological examination.

The results of serum uric acid (mg/dL) and serum creatinine (mg/dL) on day 15 and 18 are presented in Table 2. The significant differences were observed at 5% level ($p<0.05$) between the control and SBC treatment groups on day 15 and 18. The clinical signs were noted only in T_5 group (SBC-20 g/L) from the second day of gout induction (day 12) onwards, *viz.* watery droppings, dullness, depression and unthriftiness. There were no clinical signs in T_2 (SBC-5 g/L) and T_3 groups (SBC-10 g/L) till the end of trial (18 days). Whereas, only watery droppings were noticed in few birds on sixth day (day 16) of gout induction onwards in T_4 group (SBC-15 g/L). But the birds were active and feed intake was normal in T_4 group till the end of trial. There was no mortality initially for the first two days of gout induction in all groups. But mortality occurred from third day of gout induction onwards in T_5 group (SBC-20 g/L) where one bird on third day and two birds on fourth day of gout induction died. The mortality did not occur in T_2, T_3 and T_4 groups till the end of trial. The post-mortem examination of dead birds in gout induced group (SBC-20 g/L) on 13th day of trial (third day of gout induction) showed severe chalky white urate depositions on heart (Figure 1) and severe mottling of kidneys with urate deposits and dilated ureters (Figure 2). Also severe chalky white urate depositions on heart and liver leads to loss of hepatic architecture and severe enlargement and atrophy of renal lobes with dilated ureters on 14th day were noticed. These gross lesions clearly suggestive of gout lesions, hence this dosage was used for further experimental trials. Whereas the birds in other gout induction groups (T_2, T_3 and T_4) did not reveal any gross lesions at the end of experiment.

The histopathological findings revealed the disruption of cardiac myofibres and fibrous tissue infiltration in heart (Figure 3), urate crystals deposition, massive tubular necrosis and intertubular hemorrhage in kidney (Figure 4), were noted on day 14 in T_2 (gout control) group.

11th day to 14th day of age successfully induced visceral gout in broiler chicken. The results of the present study are in agreement with the Mubarak and Sharkawy (1999) [13] and Sodhi *et al.* (2008) [17]. Significant differences on mean serum uric acid values were observed between control and the treatment (SBC-20 g/L of drinking water) groups on 15th day and 18th day. The elevation of serum uric acid might induce the gout signs in SBC treated group. The uric acid values had crossed more than 20 mg/dL in 4 days of sodium bicarbonate treatment @ 20 g/L of drinking water. But the groups treated with 5 g, 10 g and 15 g/L of drinking water did not show hyperuricemia even after one week of treatment.

These findings concur with the findings of Prathapkumar *et al.* (2008) who noticed the clinical manifestation of gout in broilers only when the uric acid level crossed 20.81 mg/dL. The usage of sodium bicarbonate in drinking water is more harmful than in feed at the same concentration because broiler chicken consumes water 1.5 to 2.5 times more than feed (Julian, 1982). The high sodium causes kidney disease in broiler chicken, as it increases blood viscosity by reducing erythrocyte deformability (Mirsalimi and Julian, 1993) [12] and interferes with blood flow through capillaries in the glomerulus (Sodhi *et al*., 2008) [17]. Hence in the present study the severe reduction in glomerular filtration rates due to high sodium might have reduced urine flow and limit the renal capacities, ultimately leading to uricemia, visceral urate deposition and death (Sodhi *et al*., 2008) [17].

The serum uric acid concentration exceeded the solubility level of sodium urate which leads to urate precipitation in the body and the deposition over visceral organs. The higher uric acid in kidneys causes gout leading to development of clinical signs (Sodhi *et al*., 2008) [17] in SBC treated group.

With reference to serum creatinine level on day 15 in SBC @ 20 g/L group was 0.73±0.03 mg/dl and is in agreement with the observation of Dhara *et al.* (2010) [5] and Feizi *et al.* (2012) [7]. Whereas in other SBC treatment groups the serum creatinine concentration was not increased to a great extent to cause renal damage. Increased water consumption and watery droppings were observed in broiler chicken and these clinical signs are in line with the findings of Davison and Wideman (1992) [4] in commercial layers while spontaneously intoxicated by sodium bicarbonate. Since the higher sodium bicarbonate probably quenched the thirst by increasing sodium intake, because blood osmotic pressure in birds is a

thirst regulating factor. The bird consumed excess water because of thirst, which presumably increased feed passage rate and dilution of digestive enzymes that could cause lower nutrient digestibility leading to watery diarrhoea (Peng *et al*., 2013) [14]. Dullness, depression and unthriftiness were observed in gout affected birds and these symptoms were in agreement with findings of Jana *et al*. (2009) [9]. Mortality in SBC treatment group (20 g/L) might be due to urate crystals induced visceral organs damage (Eldaghayes *et al*., 2010) [6] which was well evidenced in gross pathology. Other treatment groups were normal without any mortality as there was no hyperuricemic condition. The gross pathological changes of dead birds in SBC @ 20 g/L group showed severe chalky white depositions over the heart and liver which leads to loss of hepatic architecture. Also severe enlargement and mottling of kidney with urate deposits and dilated ureters was noticed and these findings were in agreement with earlier reports (Mubarak and Sharkawy, 1999; Sodhi *et al.*, 2008) [13, 17]. The gout lesions might be due to precipitation of calcium sodium urate crystals in various organs particularly in kidneys and serous membranes of liver and heart (Lakkawar *et al*., 2018) [11]. The loss of hepatic architecture observed in the present study might be due to the damage induced by urate crystals. Uric acid is not toxic but when it is precipitated as crystals it can cause damage to tissues like kidneys, heart and liver (Eldaghayes *et al*., 2010) [6]. The findings of histopathology also confirms the development of gout lesions in SBC @ 20 g/L group.

Table 1: The experimental design for the induction of gout using toxic dose of sodium bicarbonate in broiler chicken

S. No.	Treatment	Group
1	T_1	Normal control
2	T_2	Sodium bicarbonate @ 5 g/L of drinking water
3	T_3	Sodium bicarbonate @ 10 g/L of drinking water
4	T_4	Sodium bicarbonate @ 15 g/L of drinking water
5	T_5	Sodium bicarbonate @ 20 g/L of drinking water

Table 2: Estimation of serum uric acid and creatinine in sodium bicarbonate toxic dose induced gout in broiler chicken (N=8), (Mean ± SE)

Parameter	Age	T_1 Control	T_2 SBC, 5 g/L	T_3 SBC, 10 g/L	T_4 SBC, 15 g/L	T_5 SBC, 20 g/L
Uric acid (mg/ dL)	Day 10	$9.89^{a}\pm0.36$	$9.76^{a}\pm0.41$	$9.75^{a}\pm0.32$	$9.69^{a}\pm0.40$	$9.80^{a}\pm0.23$
	Day 15	$9.19^{a}\pm0.23$	$10.83^{b}\pm0.67$	$11.97^{bc}\pm0.48$	$14.29^{c}\pm0.70$	$24.13^{d}\pm0.97$
	Day 18	$9.01^{a}\pm0.57$	$11.59^{b}\pm1.13$	$13.37^{bc}\pm1.50$	$16.62^{c}\pm0.69$	$28.97^{d}\pm2.31$
Creatinine (mg/ dL)	Day 10	$0.43^{a}\pm0.02$	$0.42^{a}\pm0.01$	$0.44^{a}\pm0.01$	$0.43^{a}\pm0.02$	$0.45^{ab}\pm0.00$
	Day 15	$0.42^{a}\pm0.02$	$0.51^{b}\pm0.01$	$0.54^{b}\pm0.02$	$0.58^{b}\pm0.02$	$0.73^{c}\pm0.03$
	Day 18	$0.44^{a}\pm0.02$	$0.57^{b}\pm0.01$	$0.59^{b}\pm0.03$	$0.63^{b}\pm0.04$	$0.81^{c}\pm0.02$

Columns bearing common superscript did not vary significantly at 5% (p<0.05) level

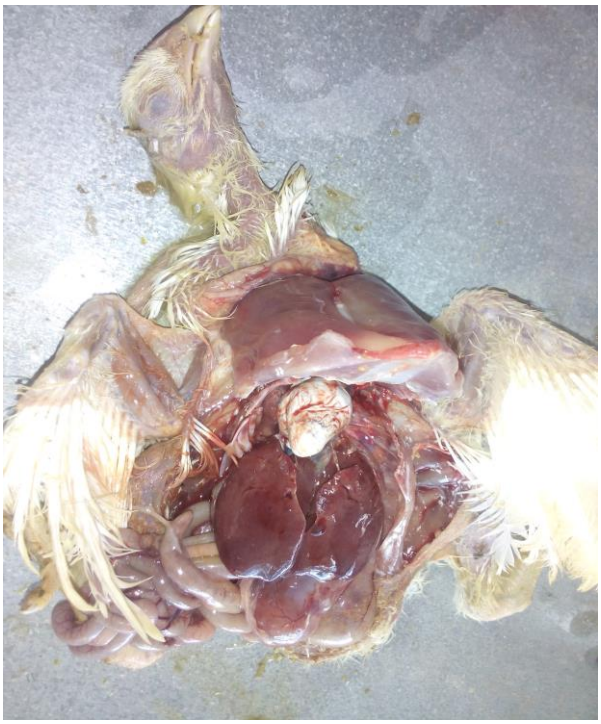

Fig 1: Severe chalky white urate deposits over heart and liver leads to loss of hepatic architecture.

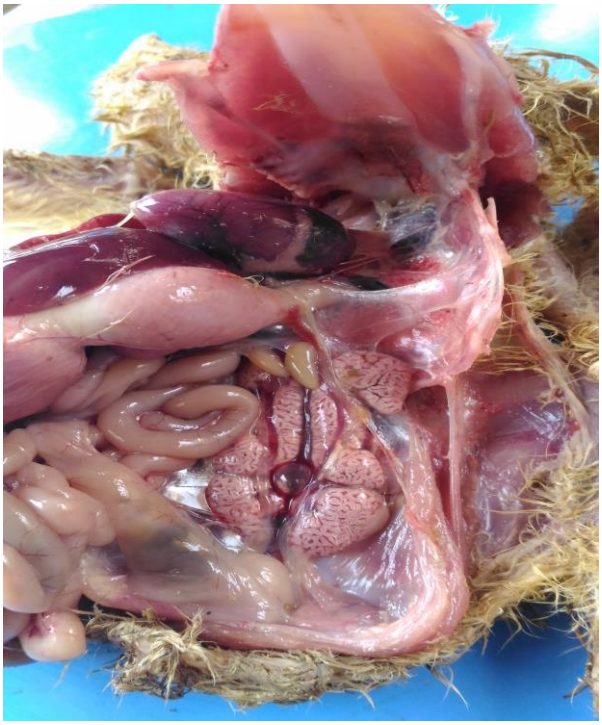

Fig 2: T_5 group-Severe mottling of kidney with urate deposits and dilated ureter.

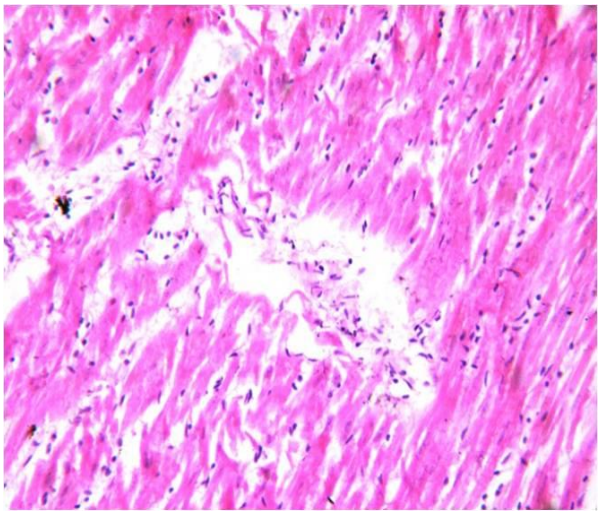

Fig 3: Disruption of cardiac myofibres and fibrous tissue infiltration in heart on day 14 after gout induction

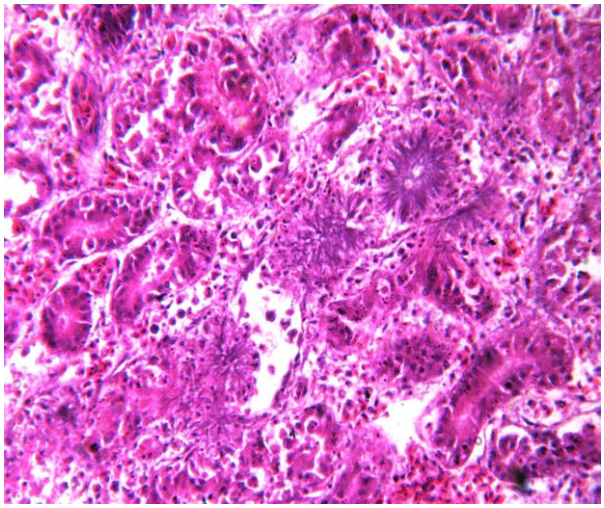

Fig 4: Urate crystals deposition, massive tubular necrosis and intertubular hemorrhage in kidney on day 14 after gout induction

Conclusion

The gout induction model was successfully standardized by administration of sodium bicarbonate @ 20 g/L in drinking water for four days from 11th day to 14th day age in broiler chicken. Hence, the scientific validation of gout induction model in the study could be useful to explore the antigout activity of new allopathy or ayurvedic drugs in broiler chicken.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

Conflict of Interest

Not available

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