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Intoxication Induced by Urea Containing Diets in Broiler Chickens: Effect on Weight Gain, Feed Conversion Ratio, Hematological and Biochemical Profiles

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Abstract

Urea as a source of cheap non-protein nitrogen is used to adulterate fish and meat meals which are basic components of broiler diets. The present study was carried out to elucidate the effects of urea on weight gain, and hematological and biochemical profiles. A total of 48 broiler chicks were randomly allotted into 4 groups, designated Groups 1, 2, 3 and 4 of 12 birds each. Birds in Groups 2, 3 and 4 were fed on diets containing urea at the levels of 1%, 2.5% and 4%, respectively. Birds in Group 1 served as control and were not exposed to urea. Experimentation period was for 3 weeks and experiment was terminated when birds were 42 days of age. Body weight of all intoxicated birds at the various intervals was significantly decreased in comparison with that of the untreated control. Compared with control, all intoxicated broilers manifested significant ($P \le 0.05$) decrease in all hematological parameters involving erythrocytic and total leucocytic counts, Hemoglobin (Hb) and Packed Cell Volume (PCV) on a dose- and time-pattern. In comparison with the control levels, biochemical profile of the intoxicated birds disclosed significant decrease in blood glucose level and significant increase in serum uric acid, urea, Alkaline Phosphatase (ALP) and Lactate Dehydrogenase (LDH) levels. Based upon the present data, it was concluded that the addition of urea to broiler diets bears serious sequences concerning the general health condition, performance, weight gain, and hematological and biochemical profiles.

Keywords

Urea, Intoxication, Broiler Chickens, Weight Gain, Feed Conversion Ratio

1. Introduction

To greatly decrease costs in poultry industry, there is a continuous search for a cheap protein source which can substitute the more expensive dietary crude protein in the regular broiler diets. In this respect, some poultry feed manufacturers implicate urea to adulterate meat and fish meals which are widely used as a convenient protein source in the broiler chicken feeds. Adulteration aims to decrease manufacturing costs through increasing the protein concentration of broiler diets by the addition of a cheap source of non-protein nitrogen (urea). Chemical analysis of urea approved its high nitrogen content which approaches 45% compared to 16% in pure protein [1] [2] [3]. In mammals, the end product of urea metabolism is ammonia which contributes to the major proportion of the resultant toxicity [4] [5] [6]. In poultry species, the main mechanisms of urea metabolic conversion are different [7] [8] [9] [10].

The published data concerning the hematological and biochemical changes induced by urea toxicity is controversial. In this respect, increased erythrocytic hematological parameters, including hemoglobin concentration and packed cell volume, and decreased total leucocytic counts were reported [11]. The mostly agreed consistent biochemical change reported in the available literature in cases of urea toxicity is the increased level of blood urea [12].

Economically, growth and nutrient utilization rates, and the final body weight of broilers are crucial factors. All efforts exerted in broiler farms are focused on achieving the highest growth rate and maximum weight gain.

The present investigation was carried out to elucidate the effect of urea intoxication on the weight gain and feed conversion ratio of broiler chickens. Also, the study aims to provide more detailed information concerning the hematological and biochemical changes which could be encountered in broiler chickens intoxicated with urea.

2. Materials and Methods

2.1. Birds

A total of 48, two-weeks-old, broiler chicks, procured from a local farm were acclimatized for one week. During the period of acclimatization, birds were kept on the proper broiler diet without addition of urea. Throughout the experimentation, all management and hygienic standards were followed. Feed and water were supplied *ad libitum* for all experimental birds.

2.2. Feed

On day 20 of age, experimental birds were shifted from broiler grower diet to the broiler finisher one. Basic composition of these diets is shown in **Table 1**.

2.3. Source of Urea

Commercially available nitrogenous fertilizer (41% nitrogen content) was used as a source of urea to be added W/W to diets of broilers.

Table 1. Basic formulation of the broiler grower and finisher diets* supplied to all experimental birds.

Ingredients	Broiler Grower Diet	Broiler Finisher Diet
Yellow Corn	63.00%	69.70%
Soy Bean	21.00%	15.00%
Meat Meal	7.00%	4.00%
Ash Meal	4.50%	3.75%
Wheat Bran	3.50%	2.50%
Limestone	0.62%	0.70%
Dicalcium Phosphate	0.01%	0.10%
Salt (NaCl)	0.16%	0.13%
Lysine	0.09%	0.12%
Methionine	0.13%	0.04%
Vitamin and Mineral Premix	0.25%	0.25%

*Int. Ibex Co., Egypt. 1) Broiler grower: 21% crude protein (crude protein with 16% nitrogen), 2.97% crude fat, 3.8% crude fibers, total metabolizable energy (calculated digestible energy) 2950 Kcl/Kg feed. 2) Broiler finisher: 19% crude protein (crude protein with 16% nitrogen), 3.85% crude fat, 2.64% crude fibers, total metabolizable energy (calculated digestible energy) 3000 Kcl/Kg feed.

2.4. Experimental Design

At 3 weeks of age, birds were randomly allotted into 4 equal groups, designated Groups 1, 2, 3, and 4, of 12 birds each. Birds in Group 1 served as control and kept on a diet free of urea, while broilers in the other 3 groups were exposed to different levels of urea. Fertilizer was added to the broiler diets at 3 different levels; 1%, 2.5% and 4% to Groups 2, 3 and 4, respectively.

All experimental birds were weighed every day to calculate the daily body weight gain, and then the average weight gain was assessed for each interval post-intoxication, *i.e.* the first, second and third week post-intoxication.

Daily feed intake was estimated, and then the average weight gain was assessed for each interval post intoxication, *i.e.* the first, second and third week post-intoxication. Based on the averages of body weight gain and feed intake at each interval, the Feed Conversion Ratio (FCR) was calculated.

FCR = feed intake (g)/body weight gain (g) [13].

All experimental birds were observed daily for performance and clinical signs.

Experimentation period extended for 3 weeks post-intoxication and the experiment was terminated when birds were 42 days of age.

2.5. Hematological and Biochemical Measurements

On days 7, 14, and 21 after feeding on diets containing urea, randomly selected 4 birds from each experimental group were weighed and blood samples were collected from wing vein into tubes with or without EDTA anti-coagulant.

Erythrocytic and total leucocytic counts were assessed using the convenient hemocytometer. Hemoglobin (Hb) concentration was measured by Cyanmet-haemoglobin methods as described previously [14] [15]. Packed Cell Volume (PCV) was estimated by a micro-hematocrit method.

For measurement of biochemical values, serum was separated from parallel blood samples. Serum level of urea was determined using a urea colorimetric assay kit (Sigma-Aldrich, UK). Uric acid level was estimated by employing a uric acid kit (Biolabo, France). Lactate Dehydrogenase (LDH) and Alkaline phosphatase (ALP) serum levels were assessed using the relevant colorimetric assay kits (Abcam, UK). All measurements were done according to the methods described by the kits manufacturers.

2.6. Statistical Analysis

All data were presented as means ± SD. The obtained data from all experimental birds were analyzed using a statistical analysis SPSS software (SPSS Inc., Chicago IL, USA). P-values ($P \le 0.05$) were considered statistically significant.

3. Results

3.1. Morbidity and Daily Observation

Starting from day 7 post-exposure to urea, all birds exposed to urea exhibited depression signs. Birds were dull, reluctant, and suffered from watery diarrhea. Severity of the signs were dose- and time-dependent, with those in 2.5%- and 4%-groups showed the most prominent signs.

On the contrary to the gradually reduced feed consumption in all groups exposed to urea, water intake of the birds in these groups was increased.

3.2. Mortality

During the third week after exposure to urea, one bird died in Group 3 (4% urea). The Bird died in a very bad health condition after manifesting the aforementioned signs.

3.3. Weight Gain

Weight gain of birds exposed to 2.5% and 4% urea was significantly lower than that of control birds. **Table 2** shows the mean averages of body weights of groups fed on diets containing urea compared to that of control group at the different intervals post intoxication. **Table 3** discloses the mean final body weights and mean carcass weights of all experimental groups at the end of experiment, *i.e.* on day 21 post-intoxication (at 42 days of age). **Figure 1** is a graphical representation for the data shown in **Table 3**.

3.4. Feed Conversion Ratio (FCR)

All intoxicated birds displayed higher feed conversion ratios at the different

Table 2. Body Weight (BW) (g) of urea intoxicated broilers at the different intervals post-intoxication compared to that of control birds.

Group	Days Post-intoxication	Body Weight
Group 1 (Control)	7	905 ± 62.217
	14	1408 ± 69.313
	21	1897 ± 81.113
Group 2 (1% Urea)	7	711 ± 53.219
	14	809* ± 43.318
	21	1060* ± 62.217
Group 3 (2.5% Urea)	7	641 ± 55.714
	14	739* ± 63.716
	21	817* ± 55.188
Group 4 (4% Urea)	7	608 ± 39.514
	14	621* ± 31.311
	21	643* ± 29.725

Values are means ± S. E. M. *Significantly different means ($P \le 0.05$).

Table 3. Final body weight (g) and carcass weight (g) of urea intoxicated groups at the end of experiment (on day 21 post-intoxication, 42 days of age) compared to that of control group.

Groups	Final Body Weight	Carcass Weight
Group 1 (Control)	1897 ± 81.113	1242 ± 70.221
Group 2 (1% Urea)	1060* ± 62.217	689* ± 57.342
Group 3 (2.5% Urea)	817* ± 55.188	519* ± 50.138
Group 4 (4% Urea)	643* ± 29.725	414* ± 26.612

Values are means ± S. E. M. *Significantly different means ($P \le 0.05$).

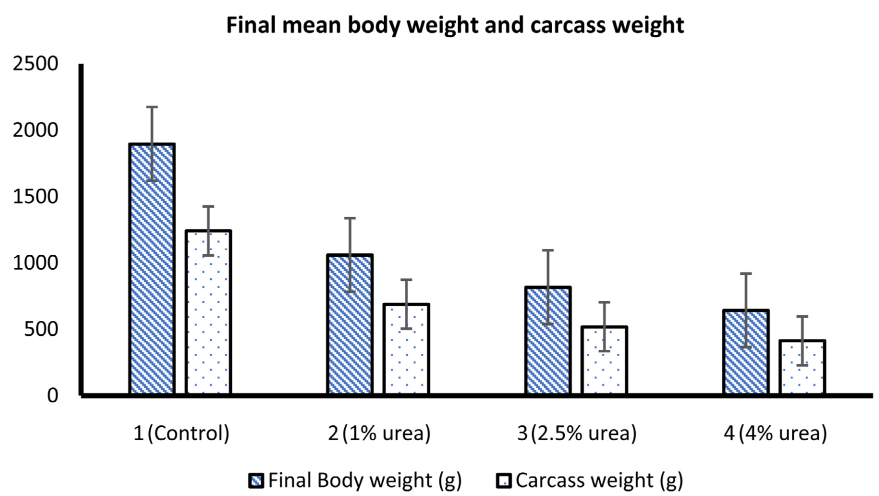


Figure 1. Final mean body weight (g) and mean carcass weight (g) at the end of experiment (on day 21 post-intoxication, 42 days of age) of urea-intoxicated groups compared to that of control group.

intervals, *i.e.* their feed intake was relatively higher compared with the control birds to gain the same unit of body weight.

Table 4 shows the daily feed intake (g), body weight gain (g) and Feed Conversion Ratio (FCR) of the urea intoxicated groups compared to control group at the different weekly intervals post-intoxication.

Figure 2 is a graphical representation of the data in Table 4. To simplify the graphical display of the data shown in Table 4, only one calculated mean average was employed to represent the 3 intervals post intoxication for each experimental group.

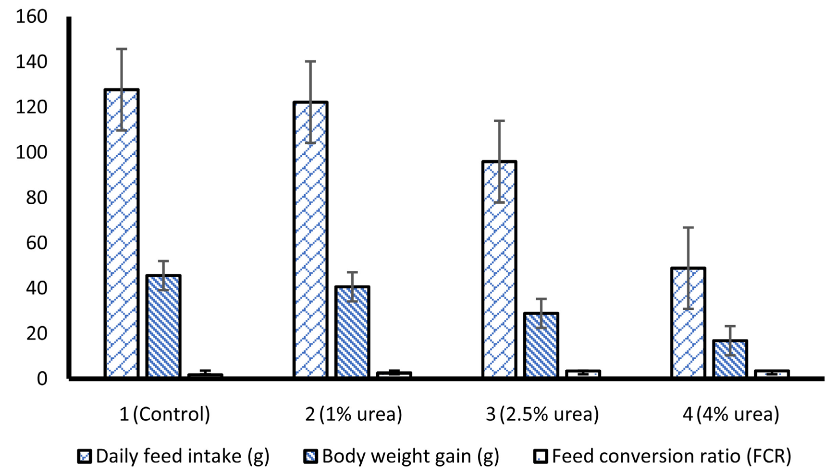


Figure 2. Feed intake (g), body weight gain (g) and Feed Conversion Ratio (FCR) of urea intoxicated groups compared to control group.

Table 4. Feed intake (g), body weight gain (g) and Feed Conversion Ratio (FCR) of urea intoxicated groups compared to control group at the different intervals post-intoxication.

Group	Weeks Post-intoxication	Feed Intake	Body Weight Gain	Feed Conversion Ratio (FCR)
Group 1 (Control)	1	117.20 ± 8.211	34.10 ± 1.871	1.81 ± 0.643
	2	128.40 ± 7.654	49.70 ± 1.764	1.86 ± 0.633
	3	137.50 ± 8.124	53.30 ± 1.802	1.93 ± 0.7.11
Group 2 (1% Urea)	1	114.60 ± 7.432	31.20 ± 1.864	2.52 ± 0.631
	2	120.30 ± 7.111	42.60 ± 1.711	2.61 ± 0.541
	3	131.80 ± 8.129	48.40 ± 1.751	2.83 ± 0.641
Group 3 (2.5%Urea)	1	109.10 ± 6.891	29.50 ± 1.642	3.40* ± 0.586
	2	97.70* ± 7.031	33.70* ± 1.631	3.51* ± 0.665
	3	81.20 ± 7.108	23.80* ± 1.644	3.73* ± 0.681
Group 4 (4% Urea)	1	80.60* ± 6.761	24.10* ± 1.428	3.43* ± 0.643
	2	43.50* ± 6.342	17.30* ± 1.463	3.57* ± 0.559
	3	22.60* ± 6.791	9.40* ± 1.474	3.77* ± 0.616

Values are means ± S. E. M. *Significantly different means ($P \le 0.05$).

3.5. Hematological and Biochemical Profiles

Hematological findings encompassed significantly decreased erythrocytic and total leucocytic counts and lowered levels of Hb and PCV in all birds exposed to urea. Decrements were dose- and time-dependent.

Biochemical parameters of broilers fed on diets containing urea involved increased serum levels of LDH and ALP, the increments were in a linear pattern with dose and time. Similarly, uric acid and urea serum levels were steadily increased with increasing urea level at the different intervals.

Table 5 and Table 6 show the various hematological and biochemical parameters assessed in the experimental groups, respectively.

Figure 3 and Figure 4 show graphical representations of hematological and biochemical changes of the intoxicated groups compared to control group. To simplify the graphical display of each hematological and biochemical parameter, the separate mean averages for the 3 intervals post intoxication for each experimental group were represented by only one calculated mean average.

4. Discussion

The focus of the current study was to elucidate the hazardous effects resulting from addition of urea to broiler diets. This is considered as a sort of adulteration by substituting the more expensive protein with a much cheaper non-protein nitrogen source. The result is increasing the dietary nitrogen to nearly 3 folds as compared with the nitrogen content of a crude protein (15%) [3].

Presently, despite the use of additive urea as a high nitrogen source, general performance of the intoxicated birds was drastically affected. Weak performance of urea exposed birds was previously report [16]. Also, body weight of all intoxicated birds at any time interval was significantly decreased if compared with the control. Decreased body weights of intoxicated broilers may be ascribed to reduced protein and energy utilization rates. From the metabolic point of view, decreased weight gain reflect a state of poor feed conversion rate and also points to the poor utilization of high nitrogen contained in urea. To make use of any dietary protein, its metabolism should be directed in the normal defined pathways [17]. Although the crude traditional protein contained in diets of the present control birds possessing much lower nitrogen, but the birds succeeded to utilize this protein properly for their growth. This was approved in the current study by the calculated daily body weight gain and Feed Conversion Ratio (FCR) which were clearly in favor of control birds. This finally reflected on a gradually increased growth rate of control broilers to reach acceptable body weight gain at the end of experiment.

Gradually decreased body weights of urea intoxicated broilers, in a dose and time manner, obviously approves the economic losses which could be resulted due to addition of dietary urea. It is worth mentioning that the economic profit of broiler farms is finally evaluated by achieving the target weight gain at the time of marketing.

Table 5. Hematological changes of urea intoxicated broilers at the different intervals post-intoxication compared with that of control birds. (a) Erythrocytic ($\times 10^6$/ml) and total leucocytic counts ($\times 10^3$/ml); (b) Hemoglobin (Hb) concentration (g/dl) and Packed Cell Volume (PCV) (%).

(a)

Groups	Days Post-intoxication	Erythrocytic Count	Total Leucocytic Count
1 (Control)	7	1.788 ± 0.051	31.17 ± 1.31
	14	1.831 ± 0.056	31.21 ± 1.79
	21	1.963 ± 0.067	34.72 ± 1.69
2 (1% Urea)	7	1.592 ± 0.112	29.27 ± 1.83
	14	1.513* ± 0.117	29.38 ± 1.61
	21	1.443* ± 0.213	28.81* ± 1.08
3 (2.5% Urea)	7	1.498* ± 0.079	28.12* ± 1.73
	14	1.389* ± 0.067	28.03* ± 1.42
	21	1.378* ± 0.097	28.1*1 ± 1.39
4 (4% Urea)	7	1.298* ± 0.048	27.72* ± 1.78
	14	1.217* ± 0.039	26.69* ± 1.87
	21	1.113* ± 0.027	26.33* ± 1.41

Values are means ± S. E. M. *Significantly different means ($P \le 0.05$).

(b)

Groups	Days Post-intoxication	Hemoglobin	Packed Cell Volume (PCV)
1 (Control)	7	8.83 ± 0.62	27.61 ± 0.02
	14	9.21 ± 0.0311	28.32 ± 0.91
	21	9.41 ± 0.029	30.27 ± 0.57
2 (1% Urea)	7	8.18 ± 0.29	26.98 ± 0.78
	14	7.79* ± 0.31	26.31 ± 0.62
	21	7.71* ± 0.49	25.97* ± 0.76
3 (2.5% Urea)	7	7.13 ± 0.31	26.03 ± 0.87
	14	6.56* ± 0.29	24.08* ± 0.61
	21	6.41* ± 0.28	23.17* ± 0.59
4 (4% Urea)	7	6.81* ± 0.613	24.09* ± 0.77
	14	5.31* ± 0.423	21.15* ± 0.62
	21	5.09* ± 0.512	20.91* ± 0.59

Values are means ± S. E. M. *Significantly different means ($P \le 0.05$).

Table 6. Biochemical changes of urea intoxicated broilers at the different intervals post-intoxication compared with that of control birds. (a) Uric acid (mg/dl), urea (mg/dl) and glucose (mg/dl) levels; (b) Alkaline Phosphatase (ALP) (U/L) and Lactate Dehydrogenase (LDH) (U/L) levels.

(a)

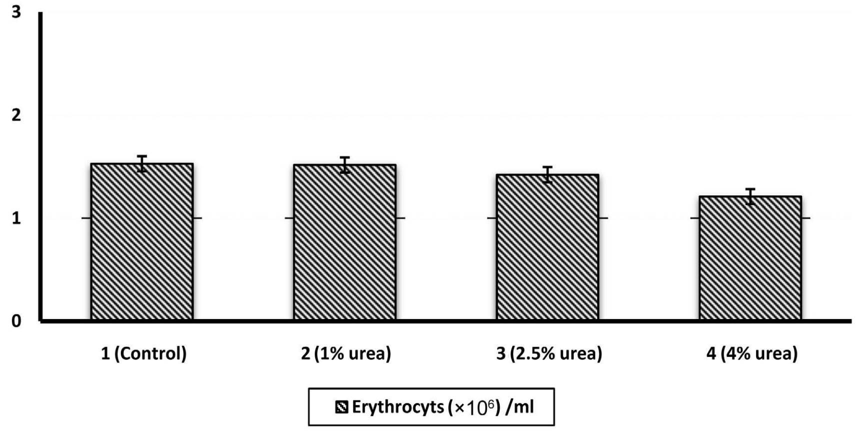
Groups	Days Post-intoxication	Uric Acid	Urea	Glucose
1 (Control)	7	10.731 ± 0.413	10.133 ± 7.626	80.012 ± 3.319
	14	12.812 ± 0.618	12.119 ± 9.636	98.722 ± 4.116
	21	14.131 ± 0.416	17.429 ± 10.813	109.823 ± 7.012
2 (1% Urea)	7	19.812* ± 1.512	39.123* ± 12.136	74.341 ± 5.118
	14	33.612* ± 1.418	96.124* ± 12.116	70.812* ± 4.815
	21	39.415* ± 1.312	167.266* ± 12.613	59.731* ± 4.213
3 (2.5%Urea)	7	24.712* ± 1.411	87.319* ± 23.122	69.243* ± 3.017
	14	37.611* ± 1.322	139.217* ± 18.191	58.316* ± 6.711
	21	42.312* ± 1.214	316.717* ± 23.128	52.313* ± 3.118
4 (4% Urea)	7	41.117* ± 1.312	192.612* ± 13.125	61.513* ± 5.612
	14	52.061* ± 1.718	411.135* ± 17.516	43.628* ± 4.119
	21	51.021* ± 1.313	488.313* ± 24.618	39.417* ± 5.683

Values are means ± S. E. M. *Significantly different means (P ≤ 0.05).

(b)

Groups	Days Post-intoxication	ALP	LDH
1 (Control)	7	88.316 ± 3.217	37.112 ± 0.216
	14	94.219 ± 4.617	41.219 ± 4.416
	21	96.724 ± 6.488	43.336 ± 3.128
2 (1% Urea)	7	90.772 ± 3.812	124.116* ± 8.118
	14	99.656 ± 7.712	213.721* ± 6.231
	21	120.468* ± 6.619	290.612* ± 4.180
3 (2.5% Urea)	7	118.119* ± 4.213	177.139* ± 8.167
	14	131.722× ± 5.518	224.216* ± 6.264
	21	139.681* ± 6.116	336.718* ± 4.418
4 (4% Urea)	7	142.112* ± 9.218	349.466* ± 5.712
	14	164.319* ± 8.127	464.319* ± 5.617
	21	189.427* ± 7.829	498.679* ± 4.616

Values are means ± S. E. M. *Significantly different means (P ≤ 0.05).



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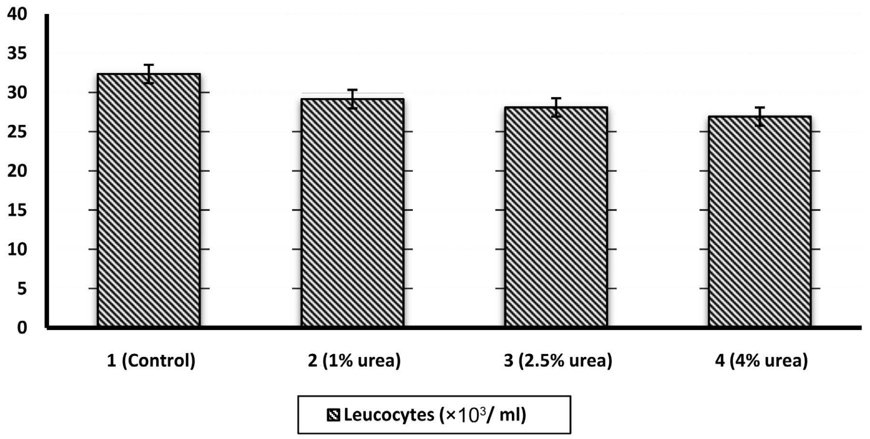

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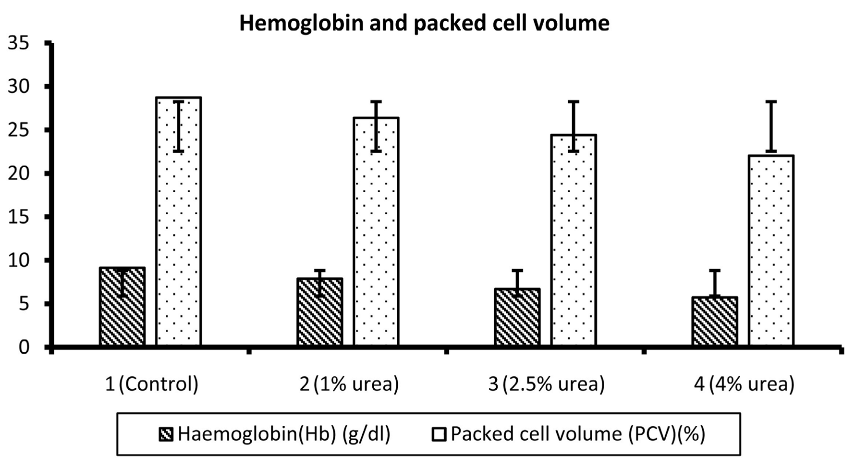

(c)

Figure 3. Hematological changes of urea intoxicated groups compared with that of control group. (a) Erythrocytic count; (b) Leucocytic count; (c) Hemoglobin and packed cell volume %.

Low blood glucose levels recorded in the presently intoxicated birds may support the possibility of decreased renal tubular reabsorption. Also, hypoglycaemia may represent a state of non-efficient metabolic activities.

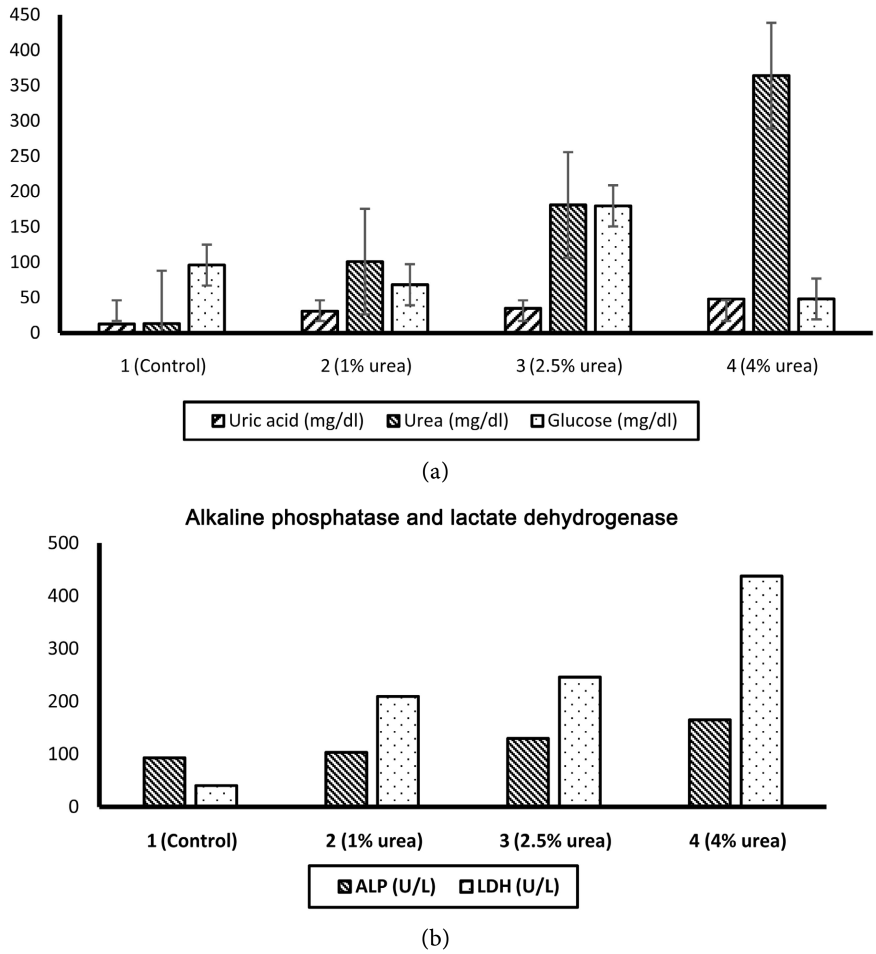

Figure 4. Biochemical changes of urea intoxicated groups compared with that of control group: (a) Uric acid (mg/dl), urea (mg/dl) and glucose (mg/dl) levels; (b) Alkaline Phosphatase (ALP) (U/L) and Lactate Dehydrogenase (LDH) (U/L) levels.

Currently, significant decrease of the various hematological erythrocytic indices, including RBCs count, Hb and PCV was recorded in all urea intoxicated birds. These diminshed erythrocytic indices might provide an evidence of a direct toxic effect of urea on bone marrow. Additionally, elevated urea and uric acid blood levels may play a worth reporting role in depressing hemopoiesis. It has been suggested that increased urea blood level may shorten life span of RBCs and thus indirectly decreases eyrthrocytic count [18]. Alternatively, erythropoietin which controls RBCs synthesis was either of low magnitude (hyposynthesis) or remarkably of low activity due to effects of urea toxicity. Erythropoietin is known to be severely affected in the presence of renal or hepatic damage [19].

It has been reported that high intake of urea is followed by rapid intestinal absorption and subsequently increased its serum level [20]. This explains the early increments of urea serum levels in the presently intoxicated birds. In mammals, urea intoxication is documented to provoke high blood urea level due to activation of hepatic ureosynthetic cycle [5]. In birds, lytic changes of urea (ureolytic activity) and the release of ammonia may occur in caecum by the aid of urease enzyme [1] [2] [20] [21] [22] [23]. However, the rapid intestinal absorption of urea possibly overcomes the ureolytic activity with ultimate steady increase of urea blood level.

Increased blood uric acid level (hyperuricaemia) in the present experimental birds is most probably the result of decreased uric acid renal excretion as a sequence of renal damage. The role of renal changes in elevation of blood urea and uric acid levels were emphasized by some reserchers [24] [25] [26]. Moreover, Dehydration due to continuous diarrhea, as observed in the currently intoxicated birds, might stimulate renal tubular reabsorption with a subsequent indirect increase of blood urea and uric acid levels [19] [24].

Our present findings encompassed significantly elevated LDH and ALP serum levels in urea intoxicated birds. Nitrogen overload as occurs in case of urea intoxication is usually associated with tissue damage [11]. Generally, when parenchymatous organs get damaged, leakage of cellular lysosomal enzymes is expected [15] [27]. Release of ALP and LDH into blood is a sensitive parameter to tissue damage [28]. Each of these released serum enzymes may reflect a particular location of tissue damage as in case of ALP level which is beneficial to express liver damage. LDH leakage and its subsequent increase in serum is more correlated with myopathic changes [15] [29].

Mortality recorded in group 3 (4% urea) is presumably attributed to multiple organ failure raised from urea intoxication.

Undoubtedly, broiler birds in the present investigation were unable to sustain the high nitrogen overload released from urea with subsequent deleterious effects. These effects were evidenced by the final obviously poor weight gain and the noticeably altered hematological and biochemical profiles of the intoxicated birds. The present data approves that urea as a substitute to dietary crude protein obviously failed to provide an equivalent nutritional value compared with regular broiler diets. Moreover, the present results indicate that addition of urea to broiler diets can lead to deterioration of the general health condition and performance of the intoxicated birds.

5. Conclusion

Conclusively, the current results indicate that supplementation of urea as a cheap source of dietary non-protein nitrogen to adulterate broiler diets bears the risk of serious consequences. Economic losses resulting from the remarkably poor weight gain of broilers at the time of marketing represent important drastic consequence.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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