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BIOCHEMICAL AND CLINICOPATHOLOGICAL STUDIES ON UROLITHIASIS IN CHICKEN BROILER BREEDERS AND COMMERCIAL LAYERS

M. Siddique and W. Hanif[1]
Department of Vet. Microbiology, University of Agriculture, Faisalabad,
[1]Poultry Research Institute, Rawalpindi, Pakistan

ABSTRACT

Wide spread problem of urolithiasis in broiler breeders and commercial layers associated with high mortality and loss of condition was investigated. Calcium and phosphorus were high in feed as well as in drinking water. Virus as a cause of urolithiasis was not ascertained. On postmortem, birds revealed visceral gout, atrophy and or hypertrophy of the kidneys. One or both ureters were packed with white uroliths figuring out as white cords. Microscopically, urate deposits along with necrosis and loss of kidney parenchyma were the predominant lesions.

INTRODUCTION

Urolithiasis and visceral gout are the conditions associated with a number of varying etiology (Niznik *et al.*, 1985; Widemen *et al.*, 1985). The losses due to urolithiasis are because of high morbidity and mortality ranging from 6 to 20 percent (Randall *et al.*, 1977). Experimentally, these conditions have been induced by excessive dietary calcium, inadequate dietary available phosphorus and nephrotopic strains of infectious bronchitis virus (Wideman *et al.*, 1983, 1985). However, the role of grey strain of IBV with respect to urolithiasis is debatable (Fitz-Coyet *et al.*, 1988; Glahn *et al.*, 1988). High calcium diet causes a significant reduction in kidney mass and a significant increase in the incidence of gross kidney damage and urolithiasis in pullets. Calcium induced kidney damage resulting into urolithiasis is reported to be reduced by supplementing high calcium diets with liquid methionine hydroxy analogue and DL-methionine (Lent and Wideman, 1993).

Various strains of chicken vary regarding their susceptibility to urolithiasis. High calcium contents (35 gm/kg) induced relatively higher kidney damage and urolithiasis in one strain of pullets than the others. Supplementing high calcium feed with 6 gm/kg DL-methionine reduced the incidence of calcium induced gross kidney damage and urolith formation in both strains. Ammonium sulfate (5.3 gm/kg) was significantly more effective than DL-methionine in reducing calcium induced kidney damage (Lent and Wideman, 1993).

Drinking water on most of the poultry farms around Faisalabad is hard (Anjum *et al.*, 1989) and this may lead to high incidence of urolithiasis. The present studies were initiated to investigate wide spread urolithiasis characterized hy high mortality and morbidity in breeder flocks in and around Rawalpindi and commercial layers around Faisalabad.

MATERIALS AND METHODS

Urolithiasis was observed in a number of broiler breeders presented for diagnosis at Poultry Development Centre, Rawalpindi and in commercial layers presented in the Department of Veterinary Microbiology, University of Agriculture, Faisalabad. Among the breeder flocks, ten affected farms having 56,000 birds, and four layer farms with 21,000 birds were selected for detailed studies. These farms were visited and detailed history of the flocks including breed, age, feed, source of drinking water and vaccination schedule was recorded (Table 1).

Samples of feed and drinking water (6 springs and 8 underground) were collected for mineral analysis. Feed samples were also analyzed for total proteins, crude fibre, total ash, crude fat and total aflatoxin levels.

Dead and sick birds were examined to record gross pathological lesions in different visceral organs. Liver and spleen from affected birds were cultured for bacteriological studies. Appropriate portions of the affected kidneys were fixed in buffered formal saline and processed for histopathological details through hematoxylin and eosin staining (Humason, 1972).

RESULTS AND DISCUSSION

Among the farms with urolithiasis, fourteen seriously affected flocks were selected for detailed investigations. In urolithiasis affected flocks there was a chronic history of emaciation and eventual death. Clinically the birds appeared depressed and dehydrated with ruffled feathers and delayed feed intake. The affected flocks presented considerable size variations in their body weights. The condition in different flocks had persisted for variable time intervals, varying from 2 to 18 weeks. Mortality in various flocks during the problematic period is presented in Table 2.

It is evident that motality in various urolithiasis affected breeder flocks varied from 3.3 to 21 percent and in layer flocks from 3 to 4.2 percent. There does not seem to be any correlation between mortality and age of the flock. All the breeder birds were maintained in a single feed but located at four different places. Various breeder flocks maintained on other commercial feeds, located in the same areas were also surveyed, but urolithiasis was not recorded in any of these flocks. This indicates that feed may be a source of urolithiasis. The affected layer flocks were provided homemix feed and in all these cases feed ingredients were procured from a single source. This showed feed as a common factor for urolithiasis. Feed as a cause of urolithiasis has been described by a number of workers (Wideman *et al.*, 1983,1985; Fitz-Coyet *et al.*, 1988; Glahn *et al.*, 1988).

These flocks were vaccinated against various prevalent diseases viz Newcastle disease, infectious bronchitis, infectious bursal disease and infectious laryngotracheitis. So there seems little role of these diseases. Infectious bronchitis (IB) virus has been reported to be one of the important aetioloigical factors for urolithiasis (Wideman *et al.*, 1985).

Although serological tests for IB could not be performed, however, all the birds had been vaccinated twice against IB indicating few chances of IB outbreaks. Moreover, neither typical clinical signs for gross and histopathological lesions of IB were recorded in the dead or sick birds. Fitz-Coyet *et al.* (1985) and Glahn *et al.* (1988) also reported urolithiasis without the involvement of IB virus.

Analysis of feed (Table 3) showed higher levels of total ash and tremendous variation particularly in the levels of calcium (Ca), phosphorus (P), sodium (Na) and potassium (K). Higher Ca and Na levels accompanied by lower P level have been reported to be among the major factors for urolithiasis (Niznik *et al.*, 1985; Wideman and Cowen, 1987). Negative potassium balance may be responsible for anal urolith formation which may be related to lower dietary levels as well as to reduce feed intake (Sillers, 1981).

Drinking water samples were analyzed for total solids, chlorides, carbonates, bicarbonates, sodium, potassium, calcium, manganese and electrical conductivity (Table 4). Although bicarbonates, sodium and calcium were found in upper limits yet all these parameters were within the permissible range. Alkaline pH of drinking water may account for mineralization. All the water samples showed moderate to higher pH. this may be regareded as one of the factors for uroliths. However, further studies are need to confirm the role of pH and salts in the urinary tract.

Table 1. Details of urolithiasis affected flocks.

Farm No.	Total birds	Breed	Age (weeks)	Week of initiation	Feed	Locality of farm
1	3000	B1	46	18	F1	Chak Shahzad, Rawalpindi
2	3000	B1	32	16	F1	Chak Shahzad, Rawalpindi
3	2000	B1	53	18	F1	Chak Shahzad, Rawalpindi
4	6000	B1	34	16	F1	Chackry Road, Rawalpindi.
5	12000	B1	42	16	F1	Chackry Road, Rawalpindi
6	13000	B1	10	2	F1	Chackry Road, Rawalpindi.
7	4000	B2	18	5	F1	Sahala, Rawalpindi
8	7000	B2	26	10	F1	Sahala, Rawalpindi
9	5000	B1	35	14	F1	Sahala, Rawalpindi
10	2000	B1	8	3	F1	Muree Road, Rawalpindi
11	4500	L1	18	2	F2	Samundri, Faisalabad.
12	3000	L2	20	4	F2	Samundri, Faisalabad.
13	5000	L2	17	5	F2	Samundri, Faisalabad.
14	8500	L2	16	4	F2	Samundri, Faisalabad
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Table 2: Mortality in various urolithiasis affected birds

Farm No.	Duration of problem (weeks)	Mortality	
		No.	Percent
1	28	220	7.3
2	16	80	4.0
3	35	65	3.3
4	18	967	16.1
5	26	2520	21.0
6	8	526	4.1
7	13	510	12.8
8	16	760	10.9
9	21	400	8.0
10	5	120	6.0
11	16	185	4.1
12	16	96	3.2
13	12	210	4.2
14	12	255	3.0

Table 3: Analysis of feed being used on various poultry farms.

Source of feed	Crude fibre (%)	Crude protein (%)	Crude fat (%)	Total mineral ash (%)	Aflatoxin (ppb)	Ca (%)	P (%)	Na (%)	K (%)
F1a	2.4	18.0	4.6	7.2	38	5.28	0.30	0.42	0.13
F1b	2.5	18.5	4.5	7.6	35	5.33	0.29	0.41	0.12
F1c	2.3	18.2	4.4	7.8	40	5.22	0.28	0.30	0.13
F1d	2.4	18.7	4.6	7.7	32	5.30	0.29	0.45	0.11
F1e	2.4	17.9	4.6	7.7	34	4.25	0.27	0.40	0.12
F1f	2.3	21.0	4.5	7.6	32	2.28	0.20	0.41	0.13
F1g	2.6	18.5	4.2	7.2	24	4.32	0.25	0.42	0.13
F1h	2.3	18.4	4.3	7.3	18	4.81	0.29	0.38	0.15
F1I	2.4	17.9	3.9	7.6	29	5.12	0.31	0.39	0.10
F1j	3.4	20.5	3.8	7.5	45	2.56	0.21	0.45	0.12
F2a	2.7	18.8	5.2	8.2	31	3.80	0.28	0.41	0.15
F2b	2.5	18.5	5.5	7.8	47	4.53	0.27	0.35	0.09
F2c	2.9	19.2	4.9	8.4	50	4.22	0.29	0.36	0.11
F2d	3.2	18.7	5.2	7.9	37	3.75	0.22	0.35	0.10

On postmortem examination, the affected carcasses were emaciated and the kidneys as well as ureters appeared mineralized. In most of the cases, both the kidneys and ureters wre affected. Uroliths in kidneys and mineralization in ureters have also been recorded by Mallinson *et al.* (1984). Heart revealed fibrosis and mineral deposits on pericardium. Mineralization of other organs was also noticed particularly in the intestinal tract and lungs. Intestinal tract appeared haemorrhagic which may be attributed to higher level of mycotoxin in the feed. Liver and spleen from the affected birds were cultured on nutrient broth, nutrient agar and blood agar but no bacterial growth was observed in any of the samples.

Histopathologically the renal tissue revealed excessive mineralization along with atrophy of surrounding tissues. All the renal tissue showed various degenerative changes and even necrosis in some of the areas. Leucocytic infiltration along with haemorrhages were also observed. At many places, kidney tissue was replaced by fibrous tissue. Similar alteration have also been recorded by Fitz-Coyet *et al.* (1988).

Urolithiasis has already been occasionally seen but with low mortality and morbidity rates. However, in these flocks there was serious illness with high mortality and morbidity. Although feed, feed ingredients and drinking water analysis revealed feed or drinking water as a source of uroliths, however, this needs further confirmation under field and experimental conditions.

Table 4: Analysis of water samples from different farms

Farm	pH	Total solids	Ec	Cl	Total hardness	CO_3	HCO_3	Na	K	Ca	Mg
1	8.10	896.51	1105.58	94.31	180.00	-	579.69	107.10	2.82	72.14	26.31
2	8.02	501.51	796.06	89.35	190.00	-	427.14	95.20	10.13	76.15	27.77
3	8.0	730.57	1159.64	148.92	130.00	-	610.20	107.10	4.52	52.10	19.00
4	8.05	850.10	1649.38	233.30	185.00	-	579.69	107.10	4.52	740.14	27.04
5	8.20	802.69	1274.12	173.74	260.00	-	610.20	107.10	2.82	104.20	38.01
6	7.94	1455.80	2310.80	615.53	405.00	-	457.65	ND	ND	162.32	59.21
7	7.82	499.51	792.88	74.46	165.00	-	305.10	ND	ND	66.13	24.12
8	7.50	1259.56	649.13	120.70	342.80	0.75	712.62	83.76	3.49	104.30	26.45
9	8.10	1456.20	807.00	175.62	203.40	-	426.00	ND	ND	81.51	20.90
10	7.95	1660.20	706.42	143.00	240.00	0.85	613.70	82.5	2.50	95.20	18.00
11	8.05	1840.50	480.16	98.22	312.00	-	714.60	70.50	ND	125.60	24.50
12	7.80	2025.00	840.00	180.40	296.50	-	816.30	ND	12.87	108.60	17.52
13	7.70	1890.50	2022.00	1820.6	264.00	1.65	415.21	72.90	13.58	98.60	27.60
14	7.92	1518.40	1615.30	1719.5	360.30	2.50	920.60	74.50	11.62	130.00	29.00

ND = Not done

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