PATHOPHYSIOLOGY OF ASCITES SYNDROME IN BROILER CHICKEN DURING WINTER UNDER LOCAL CONDITIONS

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ABSTRACT

Study on 27 broiler farms, in and around Faisalabad, during winter, revealed an overall morbidity of 0.60 per cent and mortality of 4.46 per cent. Maximum mortality observed was in birds of seven weeks of age and was 5.95 per cent. The effected birds showed clinical signs including dullness, depression, slow movements, ruffled feathers, difficult breathing and distended abdomens. Postmortem examination revealed, flabby and broad base heart, swelling of liver with smooth or dimpled surfaces, swollen and congested kidneys, congested lungs. Microscopically, heart showed degenerative and inflammatory changes, similarly, liver showed necrosis and inflammatory changes, lungs showed oedema and congestion and kidney showed congestion and degenerative changes in tubular epithelium. Serum biochemical changes included higher than normal lactic dehydrogenase (LDH) (962.62 $\pm$ 601.9 IU/L), low total proteins (4.02 $\pm$ 1.50 g/dL), albumin (1.89 $\pm$ 0.77 g/dL) and globulins (2.13 $\pm$ 0.85 g/dL) and higher sodium (891.01 $\pm$ 80.8 m.Eq./L) and potassium (78.94 $\pm$ 14.13 m.Eq./L) in affected birds. In ascitic fluids level of sodium was 189.24 $\pm$ 14.53 ppm, potassium 77.40 $\pm$ 8.70 ppm, total proteins 2.39 $\pm$ 1.30 g/dL, albumin 1.50 $\pm$ 0.77 g/dL and globulins 0.76 $\pm$ 0.58 g/dL. Level of sodium in feed and water at various poultry farms was 5766.6 $\pm$ 304.0 and 82.74 $\pm$ 21.48 ppm, respectively.

INTRODUCTION

The ascites syndrome in many low altitude countries within 15 years has been established as a common cause of deaths at farms or carcass condemnation at the processing plants (Maxwell and Robertson, 1996). This syndrome has been recognized in Pakistan (Ashfaque *et al.*, 1983). Syndrome causes losses of about $500 millions to world poultry (Maxwell and Robertson, 1996). Economic losses in Pakistan due to this syndrome have not been established but it does cause reasonably high mortality during winter and renders poultry farming expensive.

Among the causative factors, high salt intake and hypoxia are the two significant factors in producing this syndrome. In Pakistan, many ingredients used in poultry feed preparation like fish, bone, meat and feather meal contain quite high amounts of common salt used as preservative. These ingredients are used in higher proportions in winter to increase the protein concentration of feed and lead to so high salt concentration in feed that may cause toxicity (Julian *et al.*, 1992b). The underground water used at 62 per cent poultry farms in and around Faisalabad is also reported to contain high amounts of salts (Anjum *et al.*, 1989).

Low oxygen inside the shed also reported to be responsible for development of this syndrome (Maxwell *et al.*, 1990). This is likely to occur under local conditions in winter while poultry farmers try to conserve heat loss by sealing sheds with plastic curtains ultimately raising ammonia inside the sheds. The present study was undertaken to understand the pathophysiology of ascites under local conditions in winter.

MATERIALS AND METHODS

The study was carried out in winter starting from November 1996 to December 1997 including 27 broiler farms located in and around Faisalabad at an altitude of 184 meters above sea level. Ascites in dead and suspected (slaughtered) birds was confirmed at postmortem. Blood samples at the time of slaughter were collected from suspected birds (confirmed at postmortem) and serum was separated and stored at -20°C till further analysis. The ascitic fluid was also collected from the same bird and stored similarly as for serum. The feed and water samples from these farms were also collected for estimation of sodium in feed and sodium and potassium in water using flame photometer following the method described by Greenberg (1992). Five grams of feed samples were diluted with 500 mL deionized water and were digested by boiling for 15 minutes. The samples were obtained in solution form and rest of the procedure was same as for water analysis for sodium. Reading was recorded in ppm and

later converted into milli equivalents per litter.

Morbid organs were collected including liver, heart, kidneys and lungs for gross and histopathological examination and were fixed in buffered formalin. Sections were cut and stained by H&E staining methods and examined under microscope for histopathological examination.

Serum lactic dehydrogenase (LDH) was estimated by using diagnostic kit (Lab. Systems LDH-P, reagent SCE, with separate starter, product No. 950054). Total protein and albumin were estimated by biuret test as described by Levinson and Macfate (1969). Serum globulin was calculated by subtracting albumin from total proteins. Serum sodium and potassium were determined by flame photometer method as mentioned in feed and water studies.

The estimation of sodium, potassium, total proteins, albumin and globulins in peritoneal fluid was made as described for serum. The data thus obtained was analysed by using one way and two-way analysis of variance and means were compared by Tukey's test for confidence intervals by using Minitab computer software package (Anonymous, 1994).

RESULTS AND DISCUSSION

The morbidity, in overall, due to ascites assessed was less than one per cent (0.66%) and not much variation in morbidity was observed in broilers of different ages (Table 1). However, in literature, variable morbidity due to this syndrome has been reported, e.g., 1.5, 9.4, 20.8 and even 80 per cent (Julian *et al.*, 1989b; Diaz *et al.*, 1994; Al-Taweil and Kassab (1990) and Witzel *et al.*, 1990). The higher morbidity in these reports was in experimental studies. An overall mortality rate assessed was however, 4.46 per cent in present study with higher mortality in birds of older ages, i.e., five to eight weeks of age (Table 1). The overall mortality agreed with the findings of Julian *et al.* (1987) and Lopez and Suarez (1989). Similarly, Silva *et al.* (1988) and Banday and Maqbool (1992) reported mortality around 5 per cent. However, as high as 46 and 50 per cent mortalities due to this syndrome has also been reported (Samaha and El-Bassioumy, 1991; Mirsalimi *et al.*, 1993). This variation in morbidity and mortality in various reports could be due to differences in altitude level (Yersin *et al.*, 1992; Banday and Maqbool, 1992), energy level in feed (Dale and Villacres, 1988), low temperature (Lu *et al.*, 1992; Julian *et al.*, 1992a), inadequate ventilation (Enkvetchakul *et al.*, 1993), high dietary sodium (Samaha and El-Bassiouny, 1991; Julian *et al.*, 1992b) and difference in genetics of birds (Julian *et al.* 1989).

The clinical signs in affected birds were dullness, depression, slow movements, ruffled feathers, difficult breathing and distended abdomen. Almost similar signs have been reported by Fraz (1988), Calnek (1991) and Jordan (1990).

Pathophysiology of ascites

The ascites under local conditions probably occurs through hypoxia from high ammonia in sheds along with pulmonary hypertension in the presence of higher sodium serum level. The higher ammonia in sheds occurs in winter while farmers try to conserve the heat loss by sealing the sheds with plastic curtains. Higher sodium serum level (Table 2) in present study was probably due to higher sodium levels in feed and underground water (Table 4) used at various poultry farms with this syndrome. Higher sodium both in feed and water was at farms where higher aged birds were present. This probably has led to a higher per centage of mortality in birds of more than five weeks of age.

Both these factors (ammonia and sodium) in combination were probably found responsible for pulmonary changes of congestion and oedema observed during present study. This has already been reported by Julian *et al.* (1992b) and Mirsalimi *et al.* (1993) that high salt concentration in blood can lead to development of pulmonary hypertension and oedema. Similarly, Al-Taweil and Kassab (1990) and Mirsalimi *et al.* (1993) induced ascites by adding 2500 and 5000 ppm sodium in feed. Hypoxia as contributing factor in pulmonary hypertension has also been reported by Julian *et al.* (1987) and Jordan (1990) in conditions of low atmospheric oxygen or in presence of high proportion of ammonia and/or in lung diseases.

Higher level of dietary sodium has been reported to cause 30 per cent expansion in blood volume (hypervolaemia), followed by pulmonary hypertension, right ventricular failure and development of ascites in broiler chicken (Mirsalimi *et al.*, 1993). The right ventricular failure occurs after right ventricular hypertrophy. The right atrioventricular valve does not effectively seal the right atrioventricular orifice and blood pressure in the behind channels rises, i.e., in vena cava. The later had secondary effects on various organs like liver and other visceral organs with wide spread effects on vascular system. The resultant congestion of liver and obstruction to lymph return results in increased outpouring of fluid from vessels, which ultimately ends in excessive fluid accumulation

Table 1: Morbidity and mortality at various farms due to ascites in broilers of different ages.

Age (weeks)	Total No. of birds	Morbidity		Mortality	
		No.	%	No.	%
2	8500	47	0.55	148	1.74
3	1500	11	0.73	22	1.46
4	2500	27	0.11	39	2.90
5	13000	97	0.95	770	5.92
6	16300	92	0.56	587	3.60
7	22600	150	0.66	1345	5.95
8	9300	16	0.17	380	4.08
Mean	73700	62.86	0.60	470.14	4.46

Table 2: Concentration of serum lactic dehydrogenase (LDH), potassium and sodium in broilers suffering form ascites syndrome.

Age (weeks)	LDH (IU/L)	Potassium (m.Eq./L)	Sodium (m.Eq./L)
2	656.6±1050.4b	78.36±12.49b	904.8±98.8ab
3	458.7±0.10.0b	74.98±5.69b	813.5±17.9c
4	331.3±354.3b	67.84±2.14c	873.5±47.2b
5	529.4±582.6b	81.24±17.01b	928.7±44.7a
6	1597.0±550.6a	72.79±10.39b	958.7±44.7a
7	1418.3±522.1a	72.06±17.09b	918.8±77.0a
8	1747.1±254.7a	105.11±8.24a	839.3±55.1b
Mean±SD	962.6±601.9	78.94±14.13	891.0±80.8

Means with different letters in a coloumn are significantly different ($P<0.05$) from each other by Tukey's W-Honestly significant difference test.

Table 3: Concentration of serum total proteins, albumin and globulins in broilers suffering from ascites syndrome.

	Total Proteins (g/dL)	Albumin (g/dL)	Globulins (g/dL)
2	4.82±2.41a	1.95±0.93ab	2.87±1.63a
3	4.50±0.38ab	2.12±0.79ab	2.38±0.45ab
4	5.57±1.22a	2.61±0.52a	2.96±0.70a
5	4.54±2.29a	2.38±1.33a	2.17±1.11ab
6	3.32±0.46b	1.65±0.31b	1.67±0.31b
7	2.88±0.55b	1.44±0.34b	1.44±0.30b
8	2.50±0.16b	1.08±0.16b	1.42±0.10b
Means±SD	4.02±1.50	1.89±0.77	2.13±0.86

Means with different letters in a coloumn are significantly different ($P<0.05$) from each other by Tukey's W-Honestly significant difference test.

Table 4: Concentration of sodium in feed and under ground water and concentration of sodium, potassium, total proteins, albumin and globulins in peritoneal fluid in broilers of different ages suffering from ascites.

Age (weeks)	Feed Sodium	Water Sodium (ppm)	Peritoneal fluid	
			Sodium (ppm)	Potassium (ppm)
2	6133.3±81.6 a	85.08±23.92 a	194.17±14.97 ab	73.88±5.15 b
3	5000.0±00.10 b	80.00±00.10 b	185.00±00.10 c	74.00±2.83 b
4	5300.0±519.6 b	73.33±5.77 b	191.67±7.64 ab	84.00±3.61 a
5	5866.7±312.2 a	110.89±24.74 a	176.67±16.77 c	73.97±5.95 b
6	6138.9±140.9 a	72.00±16.38 b	197.22±10.93 a	77.65±5.92 b
7	5927.3±414.9 a	84.68±24.75 a	190.00±16.58 b	83.65±13.78 a
8	6000.0±00.10 a	73.25±7.42 b	190.00±14.14 b	74.75±4.60 ab
Means±SD	5766.6±304.0	82.74±21.48	189.24±14.53	77.40±8.70

	Peritoneal fluid		
	Total Proteins (g/dL)	Albumin (g/dL)	Globulins (g/dL)
2	1.335±0.07 d	0.810±0.14 c	0.523±0.17 bc
3	1.515±0.06 c	0.875±0.20 bc	0.640±0.13 b
4	2.193±0.88 b	1.393±0.40 b	0.800±0.57 a
5	2.072±1.67 b	1.478±1.04 b	0.706±0.60 b
6	3.183±1.22 a	2.153±0.76 a	0.019±0.51 c
7	2.675±1.52 b	1.553±0.85 b	1.137±0.76 a
8	3.760±0.03 a	2.256±0.16 a	1.495±0.13 a
Mean±SD	2.390±1.30	1.504±0.77	0.760±0.58

Means with different letters in a coloumn are significantly different ($P<0.05$) from each other by Tukey's W-Honestly significant difference test.

in abdominal cavity (Julian and Wilson, 1986; Julian and Goryo, 1990). The dilatation of right ventricle was also observed during present study with flabby and broad base heart appearance. Microscopic changes in heart muscles were peripheral myositis with proliferation of connective tissue and degeneration of middle muscle bundles. This has also been reported by Biswas *et al.* (1995). Cerruti-Sola *et al.* (1988) and Maxwell and Mbugua (1990) reported focal areas of myocardial degeneration and myofibril disorganization. LDH in serum during present study was also high (Table 2) as compared with values in normal chicken (636.0 IU/L; Kaneko, 1989), probably indicating muscle damage. Similarly, Maxwell *et al.* (1990) reported increase in serum LDH due to ascites in chicken. This enzyme is present in higher amounts in heart and its increase in circulation represents degeneration and necrotic changes in heart muscles (Benjamin, 1978).

The secondary effects of right ventricular failure on liver during present study included swelling and/or smooth or dimpled surfaces with severe congestion and varying degree of necrosis and fibrosis at microscopic levels. Almost similar changes have been reported by Biswas *et al.* (1995), Randall (1985) and Lu *et al.* (1992). The pathology of liver was also evident from the low total proteins and fractions in serum during present study (Table 3) as compared with normal levels reported for chicken (5.60, 2.5, and 2.88 per cent for total proteins, albumin and globulins, respectively; Kaneko, 1989).

The ascitic fluid examination during present study revealed almost half the concentration of total proteins, albumin and globulins than serum (Table 4). The lower

serum proteins were partially due to escape of serum proteins to the peritoneal fluid. Similarly, sodium and potassium were also quite high in peritoneal fluid (Table 4). This probably reflects the increased permeability of the vessels and the extent of damage to the endothelium perpetuating the ascites.

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