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# BROILER PULMONARY HYPERTENSION SYNDROME. I. INCREASED RIGHT VENTRICULAR MASS IN BROILERS EXPERIMENTALLY INFECTED WITH *AEGYPTIANELLA PULLORUM*

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ABSTRACT

HUCHZERMEYER, F. W. CILLIERS, J. A., DIAZ LAVIGNE, CELESTINA D. & BARTKOWIAK, R. A., 1987. Broiler pulmonary hypertension syndrome. I. Increased right ventricular mass in broilers experimentally infected with *Aegyptianella pullorum*. *Onderstepoort Journal of Veterinary Research*, 54, 113–114 (1987)

Infection with the obligatory intra-erythrocytic anaplasma-like rickettsia *Aegyptianella pullorum* in 4-week-old broilers at a moderate altitude of 1 200 m produced a significant increase in the mean relative right ventricular (RV) mass (RV: TV) from 0,23 in the controls to 0,31 in the infected group. This was accompanied by an increase in the number of birds suffering from severe RV hypertrophy from 14,3 % in the controls to 50 % in the infected group.

Pulmonary hypertension and subsequent RV hypertrophy could have been caused by the severe anaemia experienced in the course of the infection or by metabolic or biochemical action of *A. pullorum*. As the agent does not occur on commercial broiler farms, it cannot play a practical role in the broiler ascites syndrome.

## INTRODUCTION

Hypertrophy of the right ventricle of the heart of broilers suffering from ascites has been described by Lopez Coello, Odom & Wideman (1985), Machorro Velasco & Paasch-Martinez (1985) and Julian & Wilson (1986). The involvement in broiler ascites of a pulmonary hypertension syndrome irrespective of altitude was demonstrated by Huchzermeyer & De Ruyck (1986).

The correlation between pulmonary arterial pressure and the relative mass of the right ventricle of the heart in broiler chickens in altitude disease was demonstrated by Burton, Besch & Smith (1968) and Sillau, Cueva & Morales (1980).

The technique of dissecting the heart and the calculation of the pulmonary arterial pressure index (API) by dividing the right ventricular mass (RV) by the total ventricular mass (TV), i.e., API = RV:TV, were described by Huchzermeyer & De Ruyck (1986).

*Aegyptianella pullorum* Carpano, 1928 (*Rickettsiales, Anaplasmataceae*) transmitted by argasid ticks is an intra-erythrocytic parasite of the domestic fowl. Gothe (1967) excluded the possibility of a pre-erythrocytic phase. The course of parasitaemia during experimental infection of White Leghorn cross cockerels with *A. pullorum* and the subsequent anaemia were described by Huchzermeyer (1967).

During routine heart dissections it was noticed that some pullets, previously infected with *A. pullorum*, showed increased API values. The purpose of this study was to establish the effect of *A. pullorum* infection on the relative mass of the right ventricle of broilers.

## MATERIALS AND METHODS

### *Experimental animals*

One-day-old broiler chicks were obtained from a commercial source and reared in a heated tier brooder to the age of 4 weeks. At this age they were transferred to an open floor house and exposed to conditions prevailing during June and July in the Transvaal, cold nights (+2 °C) and warm days (+20 °C). They were fed commercial broiler starter crumbs and finisher pellets. The trial facility is situated at an approximate altitude of 1 200 m above sea level.

### *Infective material*

Chicken blood with a 5 % red blood cell infection rate from the 19th passage of the local *Aegyptianella* isolate A61/86 was collected in a heparinized tube and injected intravenously at a dosage rate of 0,1 mℓ per bird. This isolate is highly virulent. The birds were infected at the age of 4 weeks, when they were transferred to the open floor unit.

The trial group (A) consisted of 25 birds and the control group (C), which was maintained under identical conditions, of 63 birds.

### *Monitoring of the infection*

The course of the parasitaemia and the progress of the expected anaemia were monitored by preparing blood smears, counting parasites and taking microhaematocrit samples from each of 2 birds 3 times per week during the course of the trial.

### *Heart dissection*

Thirty-three days after the infection, all surviving birds of the 2 groups were slaughtered, their hearts dissected and the API values established as described by Huchzermeyer & De Ruyck (1986). Of the birds from group A that died during the trial, no API values were established with the exception of the last one, which died from ascites.

### *Statistical analysis*

Student's t-test for the analysis of 2 variates with unequal variance (Clarke, 1969), was used in the statistical analysis of the API values obtained.

## RESULTS

### *Course of infection*

The peak of parasitaemia of 15 % was reached 9 days after infection (p.i.), followed closely by severe anaemia from Day 10—15 p.i. with microhaematocrit levels as low as 13 %. From Day 10 to Day 16 9 of the trial birds died with signs of severe anaemia. One more bird from the trial group died on Day 21 p.i. with ascites and an API of 0,32. During the duration of the trial there was no mortality in the control group.

### *Relative right ventricular mass*

The arithmetical mean and standard deviations of the API values of both groups are presented in Table 1. The

TABLE 1 API values of broilers infected with *A. pullorum* (Group A) and of normal controls (Group C)

| | Group A infected | Group C controls |
|---|---|---|
| $\bar{x}$ | 0,31 | 0,23 |
| range | 0,22–0,46 | 0,14–0,40 |
| $\sigma_{n-1}$ | 0,082 | 0,055 |
| n | 16 | 63 |

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distribution of API values of both groups is shown in Table 2.

TABLE 2 Distribution of API values in % in controls and birds infected with *A. pullorum*

| | Group A infected | Group C controls |
|---|---|---|
| <0,26 | 37,5 | 73,0 |
| 0,26–0,30 | 12,5 | 12,7 |
| > 0,30 | 50,0 | 14,3 |

## DISCUSSION

The courses of parasitaemia and anaemia were similar to those described by Huchzermeyer (1967). A 36 % mortality is rather high and could possibly be attributed either to a particular susceptibility of broilers to *A. pullorum,* to the fact that the broilers in this trial were much younger than the slower growing White Leghorn cross cockerels normally used in such trials or to the fact that the strain of *Aegyptianella* used in this trial was particularly virulent. The latter had in fact become evident in preceding passages. We presumed that all 3 factors contributed to the high mortality rate in the present study.

Normal API values range from 0,14–0,25, with means in the vicinity of 0,20–0,21. Values between 0,26 and 0,30 are regarded as moderate RV hypertrophy, while values of 0,31 and more reflect a severe RV hypertrophy. In cases of ascites API values are usually above 0,30 (Huchzermeyer & De Ruyck, 1986).

Under the prevailing trial conditions of moderate altitude, pelleted commercial rations and exposure to winter temperatures in an open house, as could be expected, a number of the control birds also had elevated API values. There was, however, a significant increase ($P < 0,001$) of the mean API value of the infected group, while Table 2 shows a considerable increase in the proportion of birds with severe RV hypertrophy.

The mechanism by which this RV hypertrophy is produced by *A. pullorum* infections is not yet understood. It is possible that the severe anaemia observed in the course of the infection triggers a hypoxic reaction. It is more likely, however, that the pulmonary hypertension and the resultant RV hypertrophy were caused by the biochemistry or metabolism of *Aegyptianella*. Endotoxin of gram-negative bacteria was found to cause pulmonary hypertension by Kuida, Hinshaw, Gilbert & Visscher (1958), Tikoff, Kuida & Chiga (1966), Reeves, Daoud & Estridge (1972), Anderson, Kralios, Tsagaris & Kuida (1973) and Anderson, Tsagaris, Jubiz & Kuida (1975).

If anaemia was the triggering factor, the severe anaemia caused by a coccidial infection from *Eimeria tenella* or *necatrix,* which is much more widespread than aegyptianellosis, should have a similar effect.

Aegyptianellosis does not occur in commercial broiler flocks and consequently does not play a role in the severe losses experienced worldwide from broiler ascites as described by Julian & Wilson (1986). However, it may allow a new insight into the pathogenesis of this condition or serve as an experimental model.

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